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THE PROBLEM OF CARCINOMA OF THE LUNG*

MICHAEL E. DE BAKEY, M.D.†

Houston, Texas

Within a period of only a few decades carcinoma of the lung has assumed a position of great importance in medicine. Indeed, few conditions have engendered as much intensive interest and widespread attention as this disease in such a relatively short period of time. During these few decades its status has changed in an almost phenomenal manner from the *rarest forms of disease*[3] to one of the *commonest forms of malignancy*, being responsible now for probably as many deaths in the male population as tuberculosis. During this time, too, as a consequence of Graham's[15] pioneering demonstration of a successful resection approximately 20 years ago, a major factor in stimulating interest in the problem, it has been converted from the category of *hopeless* to that of *curable* diseases. Despite this striking ascendency in its importance, the need remains for wider appreciation of its clinical significance and a more effective program of control. This is boldly underscored by the steadily rising death rate from the disease and the pitifully small salvage rate now attainable.

The factors deserving greatest emphasis concerning this disease are its increasing occurrence and relative frequency. In almost every part of the world where the disease has been studied and the data collected and analyzed a steady and rapid rise in its occurrence has been found. Indeed, most of these studies suggest that its incidence is increasing more rapidly than any other type of cancer. In the British Empire, for example, the number of deaths from cancer of the respiratory tract increased 120 per cent during the same period in which all deaths from cancer increased only 22 per cent.[17] During the 25 year period

* Presidential Address presented during the annual assembly of The Southwestern Surgical Congress, Tuesday, October 21st, 1952, Dallas, Texas.

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ending in 1947 there was a fifteenfold increase from cancer of the lung in England[11] and during the 15 year period ending in 1945, there was a fivefold increase in its case fatality rate in Denmark.[8] In this country during the decade ending in 1948, deaths from lung cancer increased 144 per cent in contrast with an increase of only 31 per cent in deaths from all types of cancer.[23] According to studies compiled by the Metropolitan Life Insurance Company the death rate for cancer of the respiratory tract in male patients increased 30.4 per cent during the period 1946 to 1949.[4] Numerous reports from all parts of the world of analyses of institutional data based upon admission rates as well as postmortem studies have shown similar observations of an almost alarming increase in the incidence of this disease. This progressive rise has now placed carcinoma of the lung among the commonest types of malignancy, being preceded in frequency in the male population only by carcinoma of the gastrointestinal tract, skin, and prostate. Approximately 10 years ago we pointed out that in our institution it had risen in frequency among deaths coming to necropsy to exceed carcinoma of the stomach.[22] Since then similar observations have been reported from a number of other institutions.[2, 5, 14, 20, 28] At the Veterans Administration Hospital in Houston, Texas during the period 1949 to 1952, carcinoma of the lung comprised the most frequent visceral neoplasm and was more than twice as common as carcinoma of the stomach. If the present trends in the death rate from cancer of the lung continue, it may be estimated that the annual number of deaths from this disease in this country will approach a figure of almost 50,000 within the next few decades.

Although there can be no doubt of the increasing incidence of carcinoma of the lung, the question has been raised as to whether this is *real and absolute* or only *apparent and relative*. Numerous explanations have been advanced to account for this curious phenomenon, and since this phase of the subject has been reviewed previously[22] and remains speculative and controversial, its detailed consideration here would serve no useful purpose. Among the more important factors which seem to bear a significant relationship to the increasing prevalence of the disease are certain occupations and tobacco smoking. The former is exemplified by the significantly high incidence of carcinoma of the lung among workers in certain mines in which demonstrable radioactive dusts exist. A similar observation has more recently been made among workers in the chromate-producing industry.[21] Whereas these occupational factors may be of etiologic significance in effecting an increase in carcinoma of the lung among such workers, they cannot be considered responsible for the greater occurrence of the disease in the general population which is not exposed to them.

Tobacco smoking, on the other hand, has a much greater *exposure influence* because of its widespread and increasing use in the general population. For this reason and because of certain potentially carcinogenic elements in tobacco, much interest has been aroused, particularly in recent years, concerning its possible relationship to the growing incidence of carcinoma of the lung. The irritating carcinogenic effects of tobacco have long been recognized and have been demonstrated experimentally by a number of investigators. Approximately 30 years

ago Fahr[12] expressed the opinion that the rising incidence of carcinoma of the lung was due to the increase in cigarette smoking. Similar opinions were subsequently expressed by others and in our review of this phase of the subject approximately 10 years ago the evidence suggesting a causal relationship between the increasing occurrence of carcinoma of the lung and tobacco consumption was summarized.[22] In recent years there has been a revival of interest in this relationship as a result particularly of the statistical studies of Wynder and Graham[29] in this country and Doll and Hill in England.[11] According to the carefully compiled data of these investigators and their statistical analyses, highly suggestive evidence of a causal relationship between cigarette smoking and carcinoma of the lung is provided. Similar observations have also been recently reported by others.[19, 27] In a further effort to study this relationship Daff, Doll, and Kenneway[9] compiled data on the death rate of cancer of the lung and the consumption of tobacco in a number of countries in Europe, including the Balkans, over a period of about 20 years. Although there were some variations in the rate, they noted an increase in the number of cases of cancer of the lung as well as an increase in tobacco consumption in all countries. They were unable, however, to demonstrate any exact proportionality between the amount of tobacco smoked and the prevalence of cancer of the lung in different countries and at different periods. According to their data the ascent in the incidence of cancer of the lung seemed to be more rapid than that in the use of tobacco. They recognized a number of difficulties in drawing valid conclusions from such studies and emphasized the need to investigate the matter further. About all that can be said at present is that there is increasing evidence that tobacco smoking may play an etiologic role in the development of cancer of the lung and that this evidence is sufficiently suggestive to deserve further study.

The most important practical fact concerning cancer of the lung is its increasing incidence no matter whether this is real or apparent and no matter what the possible explanations may be. These latter considerations may remain speculative for some time, but for the present it is essential to recognize the fact that the disease has reached such widespread prevalence as to place it among major medical problems. The significance of this fact lies in its influence upon diagnosis, for the key to early diagnosis is a high index of suspicion. The latter, in turn, is dependent upon knowledge of the relative frequency of a condition. It is now well established that cancer of the lung is a relatively common disease and therefore deserves a high index of suspicion.

Recent analysis of our experience with carcinoma of the lung, comprising 1,122 cases, highlights a number of interesting observations, some of which have been previously presented but deserve further emphasis. In our most recent analysis the distribution by age, sex, and race is no different from that previously reported. It is predominantly a disease of the male sex, with the highest frequency in the fifth, sixth, and seventh decades of life. Although the lesion may arise in any part of the bronchial tree, analysis of our cases suggest that the frequency of the sites of involvement reflects the relative size of the various segments of the lung. Of particular interest in this connection is the

fact that in slightly more than half the cases the lesion was situated in the upper lobes and that in about one third of the cases it was situated in the periphery, an observation which has recently been made by others.[25] The significance of this lies in the fact that the diagnostic value of bronchoscopy is thus limited since such sites are usually beyond the vision of the bronchoscopist. This is reflected by the fact that a positive biopsy by means of bronchoscopy was possible in this series in only 36 per cent. Epidermoid carcinoma is by far the most frequent histologic type, occurring in a little over half the cases, and is relatively more common than other types in male patients and in the more advanced ages. The other types, undifferentiated and adenocarcinoma, however, show a greater tendency to arise more peripherally than epidermoid carcinoma. Analysis of the clinical manifestations reveals observations essentially similar to those previously presented. Of particular importance in this regard is the fact that in about one third of the cases a positive diagnosis could not be established except by exploratory thoracotomy. It is our growing conviction that as this incidence rises the salvage rate will be increased. Indeed, exploratory thoractomy provides the only means of establishing the diagnosis in the great majority of relatively early asymptomatic lesions, and these are the types that provide the best opportunity for cure.

Analysis of this series of cases also reveals a number of factors which influence prognosis and survival, including particularly the extent of the growth, the degree of malignancy, and the sex and age of the patient. Of the 1,122 patients, 43 per cent were obviously inoperable when first observed. Of the remaining 639 patients, 57 refused surgical treatment, and in 582 or 52 per cent of the entire series exploration was done. Of the latter series, 64 per cent comprising 33 per cent of the entire series were found resectable. Of particular interest is the fact that a significantly higher proportion of the lesions in the private patients were resectable than in the Charity Hospital series, the respective figures being 43 per cent and 20 per cent, thus reflecting the tendency on the part of private patients to seek medical attention earlier. The resectability rate, however, over the 17 year period comprising this experience showed little or no change in the group of private patients, the figure remaining approximately the same for this entire period. On the other hand, there has been an encouraging trend toward a higher resectability rate in the Charity Hospital group of patients. Of the 372 resected cases, 74 per cent were considered palliative resections because the tumor had already extended beyond the lung, either to involve the regional lymph nodes or contiguous structures. It is apparent from these figures that the chance of cure is relatively small, for only about one third of the entire series were found resectable, and of these only about one fourth were surgically curable. These figures clearly emphasize the need for a better appreciation of the problem and the importance of seeking better means of determining the presence of the disease at a stage when successful therapy can be applied.

Follow-up studies were obtained on all patients, and the data, calculated on the basis of the possible period of survival following operation, provide reasonably reliable figures on survival rates. The distribution curve for the survival

rate in our 372 resected cases drops rather rapidly within the first year after resection, but by the second year it begins to be stabilized and continues rather slowly downward from the third to the fifth year, the actual figure at the end of five years being 18 per cent. These data suggest that a patient who lives through the second year after operation has a good chance of being alive at the end of five years.

A comparison of these survival rates for the patients with resected and those with nonresectable lesions provides a rather striking difference. Most of the patients in the nonresectable group received some form of palliative therapy such as irradiation, nitrogen mustard, or other chemotherapeutic agents. Only 8 per cent of this group, however, were alive at the end of 1 year, as compared with 33 per cent of the resected group, and at the end of 2 years these respective figures were 2 per cent and 21 per cent. Only 1 patient in the nonresected group survived five years, whereas the survival rate in the resected group at the end of 5 years was 18 per cent.

The nature and the type of growth, particularly whether or not the lesion has extended beyond the borders of the lung, are perhaps the most significant factors affecting the prognosis. Thus, in our resected series the 5 year survival rate for those patients classified on this basis as having palliative resections, was 8 per cent, whereas the comparable rate for patients with curative resections, that is, those in which the lesion was still limited to the confines of the lung, was 41 per cent. Thus, there was a fivefold increase in the survival rate for patients with no extrapulmonary extension of the growth as compared with those in which the lesion had extended beyond the lung. This emphasizes further the need for better means of establishing the diagnosis at a stage in the growth of the lesion when it is still confined to the lung.

Thus, the gross survival rate in our entire series of 1,122 cases, at the end of 5 years, is approximately 5 per cent. In about one third of the patients, when first observed, the lesion was so far advanced that it was obviously inoperable, and only 57 per cent of the entire group were considered potentially resectable. At operation, however, only approximately two thirds of these were found resectable, thus reducing the resectability rate for the entire series to about one third. Moreover, among this relatively small number that were finally found to be resectable only about one fourth were considered surgically curable, which further reduced the salvage rate so that at the end of 5 years only about 5 per cent were alive. These figures are not much different from those recorded by many other observers whose reports have recently been summarized in another publication.[10]

The need for greater awareness of the problem and a more effective program of control of this disease is clearly demonstrated by this pitifully small salvage rate. This is further emphasized by the discouragingly long period elapsing between onset of symptoms and institution of treatment, which in our patients averaged approximately 1 year. Like others we have found that although the patient is responsible for some of this delay, a good part of it unfortunately must be attributed to the physician who first sees him. This was well emphasized by

Gibbon,[13] who observed that of the average delay of 8 months between onset of symptoms and establishment of the diagnosis, the patient was responsible for 3.5 months and the physician for 4.5 months. A somewhat similar observation was reported by Bjork,[16] who found that of the 8.4 months average duration of symptoms in his series of cases, the patient was responsible for 3.4 and the physician for 5 months. This figure can be greatly reduced and the salvage rate can be significantly increased only by wider recognition of the frequency of the disease and consequently its early consideration in any patient and especially men over 40 years of age, with unexplained chest symptoms, and by prompt institution of measures, including thoractomy, to establish the diagnosis.

Evidence is accumulating to support the belief that routine roentgenographic examination of the chest provides the most effective means of increasing the salvage rate by detection of the disease in its early asymptomatic stage, when the lesion is still localized and most favorable for cure. Roentgenography is by far the most useful diagnostic procedure for providing suggestive evidence of bronchogenic carcinoma, particularly in the early stages of the disease when there are few or even no manifestations. With increasing experience with this problem there is a growing tendency to emphasize the value of this procedure in detecting the earlier or so-called *silent* phase of the disease which may exist for months and even years before producing symptoms. The results obtained from programs of mass survey of the chest provide convincing evidence of its value in this regard. Thus, in a series of over *half a million* individuals surveyed in this manner, Scamman,[26] reported detection of carcinoma of the lung will be found at the rate of 7.8 per 100,000 persons examined and Boucot found this rate to be 21 per 100,000 in a series of 156,920 examined in this manner. On the basis of results obtained by such surveys, Hilbish[18] has estimated that carcinoma of the lung will be found at the rate of about 10 per 100,000. Others, however, report a higher figure. Thus, Guiss[16] in a compilation of 10 surveys onducted in different parts of the country and totaling 1,780,178 persons xamined, found that the incidence rate for tumor suspects averaged 0.8 per 100,000 persons examined. An even higher rate was found by Guiss in the mass chest roentgenographic survey for Los Angeles County which included 1,867,201 persons examined. The actual figure for tumor suspects in this series was 1.9 per 1,000 persons examined. The observations of Overholt and Woods[24] further emphasizes the value of this method of detecting early carcinoma of the lung. According to these observers, in 67 patients who underwent surgical exploration for tumors revealed by chest roentgenographic surveys of a total of 7,892 persons examined, 39 or 60 per cent of the tumors proved to be malignant. Of particular significance is the fact that among the patients in their series in whom carcinomas of the lung were found in the asymptomatic phase by survey roentgenograms and were promptly explored, all were resectable with no evidence of lymphatic extension in 70 per cent. On the other hand, among their cases having symptom producing cancer only 30 per cent were resectable, and only 11 per cent of these showed no lymphatic extension. Even more striking are the recent observations of Abeles and Ehrlich[1] in their report of a follow-up study of 44 patients with

single circumscribed intrathoracic densities detected by routine roentgenographic examinations. Exploratory thoracotomy was carried out in 21 of the 31 cases in this group in whom it was advised. A primary malignant lesion was found in 7 of these and in 1 the lesion was metastatic. In the remaining 10 cases in which operation was advised, it was refused by the patient in 3 and by the referring physician in 6 and deferred in 1 after a negative bronchoscopy, although later a malignant lesion was definitely established in this patient. In 5 of these 10 patients hopelessly advanced carcinoma subsequently developed. Thus, of the 44 cases originally observed, almost one third proved to be malignant, whereas in those in which exploration was advised but refused or deferred, hopelessly advanced cancer developed in one half. Many observers have stressed the importance of considering *silent* lesions especially in men over 40 years of age detected by routine chest roentgenograms as possibly malignant. This is supported by the relatively high incidence of malignancy among such lesions following surgical exploration, the reported figures ranging from 12 to 74 per cent.[10]

It is apparent that on the basis of present knowledge routine periodic roentgenographic examination of the chest provides the most effective program of control for this disease and the most effective means of increasing the salvage rate. Demonstration of an abnormal shadow by this means demands aggressive action to establish the diagnosis, and when other measures fail to reveal the exact nature of the lesion, prompt exploration is indicated. Under these circumstances the risk of exploration, except in the aged and the debilitated, may now be considered less than that involved by the consequences of delay. In light of these considerations and the frequency of carcinoma of the lung in men over 40 years of age, there is good reason to support the advocacy of routine roentgenographic examinations of the chest in this population periodically, perhaps every 6 to 12 months. It is recognized that the difficulties involved in establishing such a program of control are great but they are not insurmountable. Whereas the cost of such a program is admittedly high, this too, with proper organization and administration, could be reduced to an acceptable figure. Thus, Guiss[16] found that the unit cost of the survey in the Los Angeles County was approximately 75 cents per person and averaged 396 dollars for each chest tumor suspect. In terms of the savings of life and the conservation of the economic capacity of these individuals, this is certainly not an unreasonable price to pay. The rising death rate from cancer of the lung has now reached such proportions as to make it a public health problem fully deserving much greater measures of control than are now being applied. There is urgent reason, therefore, to organize and develop a more effective program of control directed toward early detection of this disease and based upon routine periodic roentgenographic examination of the chest in the male population over 40 years of age.

REFERENCES

1. Abeles, H., and Ehrlich, D.: Single, circumscribed intrathoracic densities, N. England J. M. *244:* 85 (Jan. 18) 1951.
2. Abrams, H. L., Spiro, R., and Goldstein, N.: Metastasis in carcinoma; analysis of 1,000 autopsied cases, Cancer *3:* 74 (Jan.) 1950.

3. Adler, I.: Primary Malignant Growths of the Lungs and Bronchi. A Pathological and Clinical Study, New York, Paul B. Hoeber, 1912.
4. Anon: The postwar cancer record. Statistical Bull. Metropolitan Life Ins. Co. *32:* 8, 1951.
5. Ariel, I. M., et al: Primary carcinoma of lung. Clinical study of 1205 cases, Cancer *3:* 229 (March) 1950.
6. Bjork, V. O.: Bronchiogenic carcinoma, Acta chir. Scandinav. (supp. 123) *95:* 1, 1947.
7. Boucot, K. R.: Mass surveys as case-finding techniques for pulmonary neoplasms, Am. Rev. Tuber. *62:* 501 (Nov.) 1950.
8. Clemmesen, J., and Busk, T.: On the apparent increase in the incidence of lung cancer in Denmark, Brit. J. Cancer, *1:* 253 (Sept.) 1947.
9. Daff, M. D., Doll, R., and Kennaway, E. L.: Cancer of the lung in relation to tobacco, Brit. J. Cancer *5:* 1 (March) 1951.
10. De Bakey, M. E., Ochsner, A., and DeCamp, P. T.: Primary carcinoma of the lung, Surgery *32:* 877–891, Nov. 1952.
11. Doll, R., and Hill, A. B.: Smoking and carcinoma of lung; preliminary report, Brit. M. J. *2:* 739 (Sept. 30) 1950.
12. Fahr: in discussion of Teutschlaender, Bronchialkrebs, Verhandl. d. deutsch. path. Gesellsch. *19:* 191, 1923.
13. Gibbon, J. H., Jr.: Cancer of lung, Tr. & Stud. Coll. Physicians, Philadelphia *17:* 49 (June) 1949.
14. Graham, E. A.: Primary cancer of the lung with special consideration of its etiology, Bull. N. York Acad. M. *27:* 261 (May) 1951.
15. Graham, E. A., and Singer, J. J.: Successful removal of entire lung for carcinoma of bronchus, J.A.M.A. *101:* 1371 (Oct. 28) 1933.
16. Guiss, L. W.: Value of mass chest roentgen-ray survey methods in control of lung cancer, Cancer *5:* 1035 (Sept.) 1952.
17. Harnett, W. L. Carcinoma of the lung. Statistical survey of 1,063 cases, Post-Grad. M. J. *19:* 33 (Feb.) 1943.
18. Hilbish, T. F.: quoted by Overholt and Schmidt, J.A.M.A. *141:* 817 (Nov. 19) 1949.
19. Levin, M. L., Goldstein, H., and Gerhardt, P. R.: Cancer and tobacco smoking: preliminary report, J.A.M.A. *143:* (May 27) 1950.
20. McSwain, B., and Williams, R. B., Jr.: The relative frequency of cancer, Cancer *4:* 416 (March) 1951.
21. Machle, W., and Gregorius, F.: Cancer of the respiratory system in the United States chromate-producing industry, Pub. Health Rep. *63:* 1114 (June) 1948.
22. Ochsner, A., and De Bakey, M. E.: Carcinoma of lung, Arch. Surg. *42:* 209 (Feb.) 1941.
23. Ochsner, A., et al: Brochiogenic carcinoma; its frequency, diagnosis and early treatment, J.A.M.A. *148:* 691 (March 1) 1952.
24. Overholt, R. H., and Woods, F. M.: Early diagnosis and treatment of cancer of the lung, N. England J. M. *245:* 555 (Oct. 11) 1951.
25. Raeburn, C.: Primary carcinoma of peripheral bronchi, Lancet *2:* 474 (Sept. 15) 1951.
26. Scaman, C. L.: Follow-up study of lung cancer suspects in mass chest x-ray survey, N. England J. M. *244:* 541 (April 12) 1951.
27. Schrek, R., et al: Tobacco smoking as an etiologic factor in disease, Cancer Res. *10:* 49 (Jan.) 1950.
28. Steiner, P. E., Butt, E. M., and Edmondson, H. A.: Pulmonary carcinoma revealed at necropsy, with reference to increasing incidence in the Los Angles County Hospital, J. Nat. Cancer Inst. *11:* 497 (Dec.) 1950.
29. Wynder, E. L., and Graham, E. A.: Tobacco smoking as possible etiologic factor in bronchogenic carcinoma; study of 684 proved cases, J.A.M.A. *143:* 329 (May 27) 1950.

SOME INTERESTING TUMORS IN THE RENAL REGION

GERSHOM J. THOMPSON, M.D.*

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The patient complaining of a swelling which he has noticed in the upper part of the abdomen presents an interesting diagnostic problem which as a rule is not difficult to solve. But the physician must be aware of the various possibilities in the realm of pathology and perhaps use his imagination a little in coming to a decision as to what has gone wrong. Since many of these masses present themselves in the flank, as well as in the upper part of the abdomen, the chance that a lesion arises primarily in the kidney requires investigation of that structure. Fortunately, modern urographic study can be made wherever there are roentgenographic facilities. The excretory urogram usually is not difficult to interpret, and I urge every physician and surgeon to sharpen his interest in this procedure. With it and with his hands he can examine the patient and often make a correct diagnosis without resorting to any more elaborate tests. Furthermore, the use of the urogram can provide information which will prevent the error of making an incision in the wrong place and thus make removal of the diseased tissue more difficult. The urogram may prove that no operation is required even though a mass exists, for there are several renal anomalies which do not require surgical intervention.

I shall not attempt to present a complete outline of all the lesions which occur but instead shall relate some interesting points which have arisen in making a diagnosis in a number of cases which I have encountered in recent years. I shall try to describe the urographic findings and other data which presented and which should have enabled any physician to make a correct diagnosis. However, I shall also point out, as might be expected, that we did not avoid preoperative diagnostic errors in all cases.

INTRINSIC RENAL LESIONS

In considering the various lesions which involve the kidney and adjacent structures it is probably best to mention intrinsic renal lesions first. Of these the following are most important.

Hypernephroma. This is the commonest form of renal tumor and may attain great size before it causes symptoms. In fact, until the tumor ulcerates into one of the calyces, hematuria does not occur. The lesion may then be only a few centimeters in diameter or may have attained a huge size.

In addition to the palpable, hard, nodular swelling which can be felt on bimanual examination to extend throughout the upper part of the abdomen and flank, the best diagnostic point is the distortion of the calyces which such a tumor produces in the urogram. Not only are the terminal minor irregularities oblit-

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erated but there may be hooklike deformity of the calyceal tips. The calyces may be pushed medially, or one of them may be greatly elongated by pressure from the tumor. If the lesion starts high in the upper pole the principal deformity may be downward displacement of the calyceal system without much actual deformity of the tips themselves. If the growth arises on the anterior surface of the kidney, it may attain a huge size without much actual deformity of the pelvis or calyces. This is also true when the growth presents directly on the posterior surface of the kidney, but in this case a lateral pyelogram made with the patient lying on his side may show forward displacement of the kidney from its ordinary position in the gutter just lateral to the spinal column. Normally, the calyceal system in the lateral view is projected directly over the spinal column.

In rare cases a perfectly normal urogram will be seen even though a large hypernephroma is present. In such cases the tumor has a very small attachment to the kidney and there is little or no pressure on the collecting system. Not long ago I saw such a patient, who gave a history of afternoon fever and of having been treated for almost a year for renal infection. I might add that fever is one of the common signs encountered with hypernephroma.

Urographic evidence of complete absence of function in the presence of hypernephroma is usually a bad prognostic sign. In these cases the renal vein is sometimes extensively involved with tumor thrombi. However, unless the general condition of the patient prohibits operation it is best to carry out surgical exploration, because the lesion may be of low grade malignancy so that careful dissection may successfully extirpate what seems to be an inoperable tumor. Rarely is operation hopeless in the absence of demonstrable metastasis in the lungs or elsewhere.

In very young adults and in children the commonest form of tumor is Wilms's tumor, but hypernephroma occasionally occurs in infancy and childhood.

Cysts. The lesion most commonly encountered is a simple cyst which cannot, in the ordinary urogram, be distinguished from the deformity caused by a solid tumor. However, when a cyst is attached to the lower border of the kidney and when the large outline projected beyond the edge is clearly visible and there is comparatively little deformity of the lower calyces, one can make the diagnosis of solitary cyst with reasonable accuracy. On palpation, such lesions are as a rule not different from hypernephroma. In some cases, however, they seem more movable. Of occasional value in interpreting the urogram is careful examination of the rest of the kidney or of the other renal shadow. If other nodular extensions on the edge of the renal shadow are visible the probabilities are that the largest lesion is a cyst. In other words, simple cyst of the kidney is often multiple or such a cyst may occur in both kidneys, one being much larger than the other.

The use of the renal arteriogram in recent years has been of value in making a differential diagnosis between solid renal tumors and simple cysts. Tumor tissue usually contains multiple, small, irregular, vascular spaces which fill when the aortogram is made, revealing puddling of medium throughout the mass.

In contrast, a cyst contains no vascular network and is recognized by the lack of puddling of the medium.

Congenital Polycystic Disease. The multiple irregularities of the calyceal tips along with the broadening of the infundibulum leading from the calyces to the renal pelvis is the best diagnostic sign of this condition. The bilaterality of the condition is well known. However, in many cases one kidney is affected to a much greater extent than the other; in fact, so much so that I have seen cases in which renal tumor was diagnosed preoperatively but on exploration congenital polycystic disease was found; following this a careful review of the urogram showed unquestionable involvement of the other kidney. Not long ago I saw a girl, 16 years of age, in whom the diagnosis of tumor of the right kidney had been made. The parents were informed of the probable diagnosis and a poor prognosis was given. The excretory urogram disclosed considerable distortion of the upper group of calyces, and the diagnosis of tumor seemed obvious. However, on exploration multiple cysts were found throughout the kidney, and subsequent review of the urogram revealed obvious deformity in the left kidney. This was one of the few cases in which we were glad to find polycystic disease, although, as you know, the prognosis in this condition is not encouraging.

When polycystic involvement is extensive, renal function may be so poor that the urographic medium is not excreted in sufficient concentration to outline the distorted calyces. In such cases the examining fingers can often detect the irregular, multiple, many-sized cysts. Such an enlargement is not seen in any other type of renal disease. If the physician finds a mass of this kind in each flank it is seldom necessary to resort to retrograde pyelography to prove the diagnosis. In fact, as a rule it is well to avoid injection of medium for it may cause a severe febrile reaction which will harm the patient.

Hematoma. This condition has been seen more frequently in recent years, perhaps because of the greater incidence of accidents, traffic and otherwise. The deformity produced in the kidney depends to some extent upon whether the rupture of renal substance has extended through the capsule or whether the bleeding has been subcapsular. The usual picture encountered is obliteration of one of the groups of calyces and temporary reduction in function of the rest of the kidney. In other words, the kidney will be best visualized in the roentgenograms made 20 minutes and 45 minutes after injection of the medium. If the bleeding is subcapsular, distortion of the calyces which approximates that seen with tumor will sometimes be present. If the bleeding is perirenal, obliteration of the margin of the psoas muscle may be seen, and if it is posterior to the kidney the calyceal system may be found displaced forward if a lateral view is obtained.

Fused Kidney. The most commonly encountered type of fused kidney is the so-called horseshoe kidney in which the isthmus extends across the spinal column, thus preventing its complete rotation during embryonic development. In the renal mass on each side the calyces point medially or anteroposteriorly rather than laterally. The ureteropelvic juncture is situated, as a rule, more lateral than is normally seen. However, not every patient with a horseshoe

kidney has this typical picture. In some cases one kidney may present in practically a normal position and the other may be situated closer to, or even directly over, the spinal column. Often this kidney is in a lower position than its mate and the calyces may appear somewhat dilated and distorted. It is in such cases that the erroneous diagnosis of tumor is sometimes made, particularly if there is complete crossed renal ectopia, all of the renal mass being transposed to the opposite side and lying below the other kidney. A number of years ago I saw such a case in which the patient was a young child who had been operated upon for acute appendicitis. The surgeon found the mass and made a diagnosis of renal tumor, thus causing great consternation in the family. An excretory urogram disclosed the true condition and fortunately both segments of the kidney functioned normally and no lesion was present. The possibility of crossed renal ectopia must always be kept in mind when the surgeon on exploration finds an unusual swelling in the abdomen. Because of the multiplicity of vascular supply, these swellings unfortunately resemble retroperitoneal tumor in many respects.

Hydronephrosis. The excretory urogram is of great value in establishing this diagnosis in the large majority of cases in which the condition occurs. The smaller lesions which are characterized by enlargement of the renal pelvis and comparatively little dilatation of the calyces are usually clearly defined in the 20 and 45 minute roentgenograms. The most frequent cause of hydronephrosis is obstruction at the ureteropelvic juncture due to anomalous insertion of the ureter or to accessory bands or vessels. However, lower ureteral obstructions caused by stricture, ureteral tumor or calculus must be kept in mind.

Another uncommon cause of hydronephrosis is the ectopic position of the ureteral orifice. Particularly is this true in cases in which there is complete duplication of the ureters. One of these may have its meatus in the urethra, in the neck of the bladder or even in the seminal vesicle. This ectopic orifice is always the one from the ureter draining the upper segment of the kidney, and if one sees an unusual enlargement of the upper pole of the kidney with apparent lateral displacement of what otherwise appears to be a normal kidney the possibility of hydronephrosis of the upper segment of a completely duplicated kidney must be suspected.

A huge hydronephrotic condition in which there is complete lack of visualization of the kidney and apparent nonfunction can sometimes be tentatively diagnosed on the basis of displacement of the intestines from one side of the abdomen to the other. The huge hydronephrotic pelvis occupies the entire side of the upper part of the abdomen and pushes the bowels clear across the spinal column. Palpation of the abdomen in such cases will disclose a huge swelling which is rather tense and, as a rule, relatively fixed and immobile.

Leukemic Infiltration. This is a comparatively rare condition in which there is apparent intrinsic deformity of the kidney due to infiltration of myelocytes. It is part of the general picture of leukemia and causes distortion of the kidneys not unlike that seen with congenital polycystic disease. The prognosis in such conditions is extremely poor.

Other intrinsic lesions, which involve the kidney comparatively rarely and hence need not be discussed here, are Echinococcus disease, metastatic malignant growths from other organs, and mixed tumors in both children and adults. I might also mention papillary epithelioma of the renal pelvis. In this condition, transitional cell or squamous cell carcinoma arises in one of the calyces and obliterates it, or if the growth develops in the renal pelvis an irregular filling defect is noted in the urogram. No palpable mass develops unless the intrapelvic growth obstructs the outflow of urine and causes hydronephrosis.

PERIRENAL LESIONS

The great value of the excretory urogram in diagnosing perirenal lesions becomes more apparent as one's experience with this diagnostic method increases. Among the more commonly encountered perirenal lesions are the following:

Lipomas and Myxomatous Tumors. Great displacement of the kidney can result from these tumors. These growths, which arise in the retroperitoneal fatty tissue, are usually painless and attain a very large size before the patient presents himself for examination. The urogram reveals great displacement of a kidney which otherwise appears normal. There is no distortion of the calyces or pelvis, and the only abnormality is the position of the kidney. In some cases the kidney is displaced clear across the spinal column and superimposed over the opposite kidney. Some lengthening of the renal pedicle must occur in such cases. Surgical removal of these tumors, while laborious and time consuming, is often gratifying. Many pounds of fatty tumor sometimes containing fibrous elements can be removed and it is possible to restore the kidney to its normal position. Postoperative urograms reveal what seems to be a perfectly normal appearance, the kidney being none the worse for its previous marked displacement.

Adrenal Tumors. These tumors, when small, do not cause renal displacement. However, the suspicion of an adrenal tumor sometimes arises when one kidney is found lying at a much lower level than the other kidney. The use of perirenal insufflation has been suggested in helping make a diagnosis when these lesions are small. The outline of the enlarged adrenal gland can sometimes be well brought out by this method. A few years ago my colleagues and I saw a patient in whom a diagnosis of slight right renal ptosis had been made six years earlier. Because comparatively little if any discomfort was suffered, no treatment was advised. However, at the time of her second admission she complained of amenorrhea, and reexamination disclosed the previously diagnosed ptosed kidney to be pushed much lower than formerly. A diagnosis of adrenal tumor was made and on exploration a large tumor of this structure was found and removed. Fortunately, the tumor was of a low grade malignancy, and the patient has remained well for the past several years.

Involvement of the Retroperitoneal Lymph Nodes. Involvement of the retroperitoneal lymphatics sometimes causes lateral displacement of the lower pole of the kidney and lateral displacement of the ureter. If the condition is unilateral, one must suspect tumor of the testicle as the possible cause. It is often surprising

to find a huge involvement of the retroperitoneal lymph nodes and a tumor of small size in the testicle—the tumor may be, in fact, only a centimeter or 2 in diameter. When displacement of the lower poles of the kidneys and displacement of the ureters occur bilaterally, generalized lymphadenopathy usually is found. I have seen this condition occur in a patient with lymphocytic leukemia. Here, too, the prognosis is extremely grave.

Perinephritic Abscess. From the urographic standpoint the picture most frequently seen with perinephritic abscess is slight displacement of the kidney forward and obliteration of the margin of the psoas muscle. The pressure of the collected fluid behind the renal pelvis may cause sufficient thinning of the medium to give the appearance of a filling defect in the renal pelvis. The presence of other findings such as fever, acute tenderness, and a high leukocyte count aids in the diagnosis of this condition in contrast to tumor.

Psoas Abscess. An infrequently encountered perirenal lesion which causes displacement of the lower pole of the kidney and the ureter is psoas abscess. The huge swelling from such cause sometimes pushes the lower pole of the kidney 6 or 8 cm. laterally. The presence of the abscess may be suspected because of the fact that the lateral margin of the psoas muscle can be followed from its highest point of origin down along the edge of the huge swelling. Sometimes, bony spurs or other deformity of one of the vertebrae is also a lead in making the diagnosis. A very large swelling, usually unilateral but sometimes bilateral and lying adjacent to the spinal column, can be palpated. After drainage of such an abscess the kidney and ureter return to a normal position.

TUMORS OF ADJACENT ORGANS

The excretory urogram is often useful in exclusion of the kidneys as the source of trouble when one suspects disease in organs which lie adjacent to them. The localization of a mass in some other structure can often be more accurately determined if one is certain that the kidneys are normal. This is particularly true when one suspects enlargement of the spleen. While an enlarged spleen has a rather typical feel and often one can palpate the splenic notches, there are times, particularly in obese patients, when some indecision exists and a urogram is reassuring. The urogram usually shows that the left kidney is functioning well and in normal position or perhaps only slightly displaced downward and medially by the enlarged spleen.

Pancreatic Lesions. The pancreas is sometimes the seat of trouble when a mass is present in the left upper part of the abdomen. I recently examined a middle-aged woman who complained of a swelling at this site, and an excretory urogram disclosed what appeared to be a cyst attached to the lower pole of the left kidney. The mass was easily palpable and freely movable and it seemed to extend from the front of the abdomen through into the flank. It moved readily on respiration, and in view of a definite deformity of the lower calyces it seemed a certainty that the mass was a large simple cyst of the kidney. However, renal exploration revealed that the kidney was in normal condition and that the cyst arose in the tail of the pancreas. With some difficulty the large cyst was dissected out and the

resulting defect closed. In retrospect, a retrograde left pyelogram would undoubtedly have excluded the kidney as the primary source of trouble. The operation was a little more difficult because the incision was placed in the lumbar region rather than anteriorly.

Another interesting pancreatic tumor was observed recently in a middle-aged man whose only complaint was severe pain in the left testicle. He gave a history of having passed stones from the kidneys on several occasions, and an excretory urogram revealed a nonfunctioning left kidney with a small calculus present in the lower calyces of the right kidney which was functioning normally. Catheterization of the left ureter and retrograde pyelograms disclosed obstruction in the upper third of the ureter just below the ureteropelvic juncture. A diagnosis of nonopaque calculus was made, and at operation, instead of a stone, carcinoma of the tail of the pancreas with involvement of the ureter adjacent to it was found. The moderately hydronephrotic left kidney was removed and subsequently the patient received roentgenotherapy. We have seen several other cases of this type in which pancreatic lesions directed suspicion at the left kidney and ureter.

Cysts of the Liver. A large hepatic lobule sometimes extends down adjacent to and partially overlies the right kidney in such a way that the readily palpable mass seems to arise in the kidney itself. This condition cannot be regarded as abnormal, but when the mass is actually a cyst replacing the hepatic tissue such as was the case in a patient observed recently, considerable confusion may arise and the kidney may be erroneously considered as the primary source of the cyst. In one instance the cyst caused such unusual pressure on the lower pole of the kidney that it prevented filling of the lower calyces and it seemed certain that there was an intrinsic renal lesion. Retrograde pyelographic study was not made. On exploration a cyst 10 cm. in diameter replacing one of the lobes of the liver was found, the kidney itself being entirely normal. The cyst was excised and the resulting raw surface in the liver closed by suture. Postoperative urograms showed that the right kidney was quite normal.

Gallbladder. It is rare indeed that the gallbladder, even though greatly distended, presents a picture which will cause confusion in the interpretation of the excretory urogram. Gallstones, because of their unusual shape and often facetted appearance, are practically never mistaken for renal calculi. However, in a recently observed case, what appeared to be a large tumor in the renal region proved on exploration to be hydrops of the gallbladder. The hugely distended gallbladder had pressed so closely on the kidney that it seemed to be actually a part of that structure. A flank incision was made but fortunately this gave excellent exposure of the gallbladder and its ducts and removal of the diseased structure was easily accomplished. In retrospect, examination of the urogram disclosed very little deformity of the calyces, and furthermore, the outline of the kidney could be seen clearly through the overlying cystic-appearing structure. Preoperatively, suspicion should have been directed at the gallbladder rather than the kidney.

COMMENT

The above resume is not intended to be a complete review of all possible lesions that occur in and about the kidneys. It is intended merely to call attention to some of the interesting lesions that occur and to suggest that general surgeons can make use of the excretory urogram as an aid in differential diagnosis. Urographic study should precede gastrointestinal roentgenography because barium obscures the renal areas and accurate study cannot then be done until the stomach and intestines are completely cleared of the retained barium. This obvious fact is often overlooked. Excretory urographic study suffices in the great majority of cases, and in only a small percentage of patients observed is retrograde pyelographic study needed to clarify the diagnosis. Use of the urogram will enable the general surgeon to diagnose primary urologic disorders in many cases in which symptoms seem, on first consideration, to be of a gastrointestinal nature. The slight delay involved by resorting to urography is often very worth while, and in any case it does not interfere with subsequent gastrointestinal roentgenographic investigation.

SUMMARY

More widespread use of the intravenous or excretory urogram should be made. With it the general surgeon can often obtain information of great diagnostic value. Careful survey of the urogram often enables the physician and surgeon to avoid operative errors or to prove that surgical treatment is unnecessary for many of the enlargements which are found in the region of the kidneys. Fewer exploratory operations will be necessary if urographic study is included as part of the routine examination of the patient who has a mass in the abdomen.

OSTEOID OSTEOMA OF THE LAMINA AND ITS TREATMENT

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The clinical pathologic entity of osteoid osteoma was first described by Jaffe in 1935.[28] It is a small oval benign neoplasm of the bone. When situated in a vertebral lamina it can well mirror the clinical features of a spinal nerve root tumor. It is for this reason that I wish to report such a case and the specific surgical treatment.

There are 217 instances of this tumor reported in the English literature 19 of which were in the vertebral column.[1-66] The lamina was involved in 2 patients.

The patients have been almost entirely adolescents or young adults with the greatest incidence in the second decade of life (Table I). It occurs more than twice as frequently in males as females. Most any bone may be involved although there are no reported instances of cranial involvement. (Table II and III).

The principal symptom is localized pain in the involved region. The pain gradually increases in severity and seems to be more severe at night. It can be consistently relieved with salicylates. There are no febrile episodes even though there may be some swelling of the surrounding soft tissues. The duration of symptoms in the majority of patients varies from a few months to a few years. The most important physical finding is localized point tenderness over the site of the lesion. There may be swelling and occasionally redness. Roentgenograms of the affected bone show a small oval or round relatively radiolucent or radiopaque area. In the early stage of evolution, the lesion tends to produce a relatively radiopaque shadow. If fully developed, Jaffe believes that it appears as a relatively radiolucent or oval area surrounded by a zone of radiopacity.[29] Frequently roentgen rays fail to reveal such a lesion unless an overexposed technic is employed. It may be also impossible to demonstrate a lesion in the vertebral column due to its small size and numerous overlying structures. Therefore, the diagnosis is made by the triad: localized pain, focal tenderness, and characteristic roentgenologic findings in the absence of inflammatory disease.

The pathologic lesion as defined by Jaffe is one that usually does not exceed a centimeter in its greatest dimension and that it may lie within the spongiosa, or against the inner surface of bone cortex, or even within the cortex.[29] Although the nidus-like focus is osteoid osteoma *per se*, the total abnormal area of affected bone may be very large due to a perifocal zone of bone thickening or sclerosis. The lesion grossly appears as a blood-stained, gritty, friable, cancellus-like bone. The cut surface shows reddish brown flecks mixed with a pearl gray material. The reddish brown flecks represent a calcified osteoma quite similar to callus and the pearl gray represents the osteoid portion. Jaffe believes this is a slow

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growing benign osteogenic tumor.[30] Initially there is a proliferation of local mesenchymal tissue, particularly osteoblasts. Later, a considerable amount of intercellular substance is found between the osteoblasts. This intercellular

TABLE I

Ages of patients with osteoid osteoma

0–5 years	10
6–10 years	22
11–15 years	60
16–20 years	56*
21–25 years	36
26–30 years	16
31–35 years	8
36 and over	7
Ages not given	3
Number of cases	218

* Including author's patient.

TABLE II

Sites of osteoid osteoma

Tibia	60
Femur	52
Vertebra	20*
Humerus	16
Astragalus	11
Phalanges—manual	10
Phalanges—pedal	9
Calcaneus	8
Fibula	8
Radius	5
Ulna	5
Iliac bone	3
Navicular—carpal	2
Navicular—tarsal	2
Ankle region (Site not given precisely)	1
Mandible	1
Metatarsal	1
Patella	1
Pubis	1
Rib	1
Scapula	1
Number of cases	218

* Including author's patient.

substance slowly becomes calcified. When the tumor is fully developed, there are compact trabeculae of hypercalcified bone, and cellular and vascular intertrabecular tissue. Finally, the osteoma aspect is more prominent microscopically

than the osteoid aspect. There have been many attempts to demonstrate evidence of an inflammatory process to account for this lesion, but without success.

The only satisfactory treatment for such tumors is surgical excision. The symptoms promptly disappear after complete removal.

CASE REPORT

An 18 year old white man was referred by Dr. Charles Adkins of Beaumont, Texas. His complaint, of two years duration, was pain in the lower thoracic region just to the right of the midline with sharp radicular pain along the right costal margin to the midaxillary line. The pain had gradually increased in severity. Coughing, straining, and sneezing increased the pain, especially the radicular aspect. He developed a scoliosis during the past year. The pain was aggravated at night but was relieved temporarily by Alka Seltzer which contains salicylates. There was no history of trauma or inflammatory disease.

TABLE III

Vertebral column involvement by osteoid osteoma

Lamina	3
(1) Superior surface of lamina of vertebra C-6	
(2) Right lamina of vertebra L-2	
(3) Cephalic portion of lamina of D-8*	
Pedicle	2
(1) Left pedicle and adjacent parts of vertebra C-2	
(2) Pedicle of vertebra L-3	
Spinous process	2
(1) Spinous process of vertebra C-4	
(2) Spinous process of vertebra L-1	
Transverse process	2
(1) "Probably left transverse process" of vertebra L-1	
(2) Right transverse process of vertebra L-1	
Other site	1
(1) Right small posterior articulation between 10th and 11th thoracic vertebrae	
Cases in which locations in vertebrae were not given	10
Total cases	20

* Including author's patient.

This patient was admitted to the neurosurgical service of the John Sealy Hospital on May 4, 1951. He had a moderate dorsal scoliosis with the concavity to the right. There was marked point tenderness over the eighth dorsal spine. No other significant findings in either the general or the neurological examination were noted. Roentgenograms of the thoracic spine taken in the anteriorposterior, lateral, and oblique views were considered normal except for the scoliosis. The only abnormality in the cerebrospinal fluid reported was a 61 mg. per cent of total protein (50 mg. per cent is considered the upper limits of normal). A pantopaque myelogram revealed a constant small extradural filling defect on the right side at the level of the ninth dorsal vertebral body. The patient was discharged from the hospital in order that he might complete the school year and graduate from high school. He was readmitted to the hospital on July 15, 1951 with a diagnosis of a perineural fibroblastoma of the eighth dorsal nerve root. Another cerebrospinal fluid examination showed a total protein of 64 mg. per cent. On July 18, a right dorsal hemilaminectomy from the eighth through the tenth laminae was done. A well circumscribed raised reddish lesion was found at the lateral cephalic portion of the eighth dorsal lamina. The lesion measured

about 1½ cm. in its greatest diameter. It was very soft and friable. A complete removal was easily done using a curette. The dural surface of the lamina had not been perforated by the growth. The nerve root was angulated as it entered its intervertebral foramen which appeared to be narrowed. The edges of the foramen were thickened suggesting some reactive changes. The foramen was therefore unroofed to give the nerve root more room. The dura was opened and no abnormalities of the exposed spinal cord and nerve roots at these three levels were noted. The wound was closed in layers with interrupted sutures.

The patient's postoperative course was uneventful and he was discharged asymptomatic. He has remained free from pain to date (2 years postoperative).

Pathologist's Report

Specimens of tissue were sent to Dr. John Childers, Surgical Pathologist at John Sealy Hospital, and he reported the following: "Sections of bone from the eighth dorsal lamina show a variety of histologic changes. In some areas the marrow spaces contain fibrous and adipose tissue with a few lymphocytes and mononuclear cells scattered throughout. In

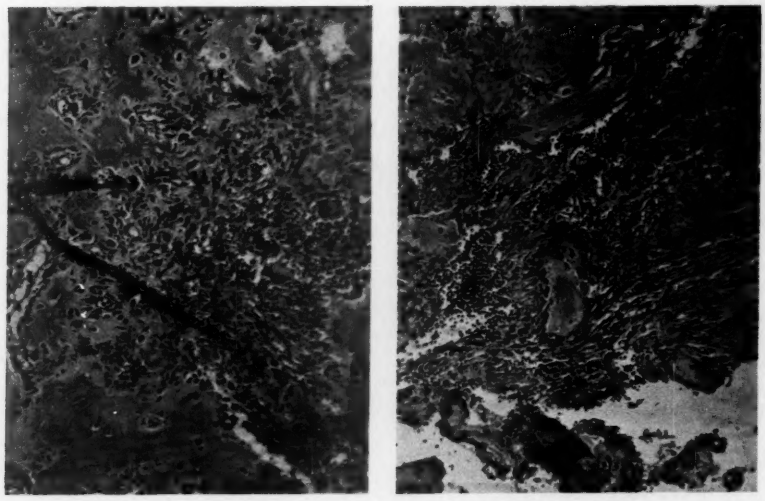

FIG. 1. Photomicrographs of the osteoma removed from the eighth dorsal lamina

other areas the normal trabecular architecture is replaced by a dense irregular network of osteoid tissue. In one fragment there is a fairly uniform junction between normal bony trabeculae and osteoid tissue. The spaces between islands of mottled osteoid tissue contain numerous vascular channels, osteoblasts, osteoclasts, and a few mononuclear cells with foamy cytoplasm. At the periphery of a section containing normal trabeculae, there is a dense arrangement of osseous tissue in which the osteocyts are incorporated in the mottled staining matrix in a very irregular manner (fig. 1). The final interpretation was osteoid osteoma of the eighth thoracic lamina.

DISCUSSION

The variety of symptoms and findings associated with vertebral column osteoid osteoma have for the most part depended upon the involved level. Such observations as wryneck, scoliosis, muscle spasm, point tenderness, lumbar lordosis, pain referred to nearby joints, and limitation of motion have been

recorded. There have been published previously reports on 19 patients with osteoid osteoma involving the vertebral column, but unfortunately not all of the reports stated exactly which parts of the vertebrae were involved. Sobel,[59] in a case report in which a cervical lamina was involved stated that 8 cases of vertebral osteoid osteomas had been reported previously and that only the neural arch had been involved. He stated that 1 patient with an osteoid osteoma of the vertebral body was observed by Jaffe immediately before his publication and that this was the only known case with such involvement. We were unable to locate any reports of cases with this bony lesion of the vertebral body. Sherman, in referring to other published accounts of this entity, noted that when the vertebral column was attacked there might be nerve root or spinal cord symptomatology.[59] Pritchard and McKay[56] reported a case in which the patient had radiating pain in the costovertebral angle to the flank caused by an osteoid osteoma of the transverse process of the first lumbar vertebra.

Sherman had observed that, in the presence of an osteoid osteoma, there were often hypertrophic changes in nearby joints.[58] When the vertebral column was involved, the small joints were often affected. The patient that we have presented in this report had such joint changes.

The associated hypertrophic joint changes in osteoid osteoma of the lamina are probably a very important factor in the production of the clinical signs and symptoms. It would seem that these changes are actually responsible for the radicular pain rather than the tumor itself. In the operative findings described here it was actually the foramen constriction and angulation of the nerve root that produced the symptoms causing the patient to seek relief. Had the laminal tumor been responsible, one might have expected spinal cord symptomatology or just thoracic pain. We believe that this concept is important in the surgical treatment of such lesions. One might postulate that had only the osteoma itself been removed without the foraminotomy, relief of the radicular pain would not have been as immediate and complete.

The preoperative differential diagnosis might cause some concern. A laminal osteoid osteoma can simulate a nerve root tumor in symptomatology, cerebrospinal fluid findings, and physical findings. However, it would seem that at the time of operation it would be most difficult to overlook if one inspected the nearby lamina in detail. The absence of roentgenologic findings at the site of the tumor makes a preoperative diagnosis difficult, unless an overexposed roentgenologic technic is used.

SUMMARY

Osteoid osteomas of the lamina can simulate nerve root tumors. We believe that the surgical treatment of such lesions involves more than the simple removal of the tumor. Since there is often an associated hypertrophic joint change in the vicinity of the tumor, foraminotomy of the affected nerve should be done. A brief review of the literature with a case report is presented.

REFERENCES

1. Barron, L. J.: Osteoid-osteoma of right os calcis; case report, Bull. Hosp. Joint Dis. *3:* 141 (Oct.) 1942.

2. Brailsford, J. F.: Importance of radiology in early diagnosis of bone tumors, Brit. J. Radiol. *10:* 171 (March) 1937.
3. Brailsford, J. F.: Chronic sub-periosteal abscess, Brit. J. Radiol. *15:* 313 (Nov.) 1942.
4. Brailsford, J. F.: Brodie's abscess and its differential diagnosis, Brit. M. J. *2:* 119 (July) 1938.
5. Brailsford, J. F., Scarff, R. W., and Ogilvie, W. H.: Discussion on malignant tumors of bone, Proc. Roy. Soc. Med. *30:* 781 (April) 1937.
6. Brailsford, J. F.: Radiological evidence of malignancy in bone tumors, and its relation to biopsy, Radiology *33:* 476 (Oct.) 1939.
7. Brown, R. C., and Ghormley, R. K.: Solitary eccentric (cortical) abscess in bone, Surgery *14:* 541 (Oct.) 1943.
8. Campos, O. P.: Osteoid-osteoma of cervical spinous process; report of case, J. Internat. Coll. Surgeons *9:* 112 (Jan.-Feb.) 1946.
9. Chandler, F. A., and Kaell, H. I.: Osteoid-osteoma, Arch. Surg. *60:* 294 (Feb.) 1950.
10. Cohen, L. J., and Van Brown, D.: Osteoid-osteoma; case report, Surgery *28:* 751 (Oct.) 1950.
11. Coley, B. L.: Neoplasms of Bone, New York, Paul B. Hoeber, Inc. 765, 1949.
12. Coley, B. L., and Lenson, N.: Osteoid osteoma, Am. J. Surg. *77:* 3 (Jan.) 1949.
13. Compere, E. L.: Streptococcus viridans osteomyelitis, J. Bone & Joint Surg. *14:* 244 (April) 1932.
14. Dockerty, M. B., Ghormley, R. K., and Jackson, A. E.: Osteoid osteoma; a clinicopathologic study of 20 cases, Ann. Surg. *133:* 77 (Jan.) 1951.
15. Dockerty, M. B., Ghormley, R. K., and Jackson, A. E.: Osteoid osteoma: clinical study of 20 cases, Proc. Staff Meet., Mayo Clin. *24:* 380 (July 6) 1949.
16. Furedi, A.: Study of the so called Osteofibroma of maxilla, The Dental Cosmos *77:* 999, 1935.
17. Geshickter, C. F., and Copeland, M. M.: Tumors of Bone; (Revised Edition) New York: Amer. Journal of Cancer, 1936.
18. Goldenberg, R. R.: Osteoid-osteoma, J. M. Soc. New Jersey *45:* 104 (March) 1948.
19. Hamilton, J. F.: Osteoid osteoma; with case reports, Surg., Gynec. & Obst. (Nov.) 1945.
20. Hamilton, J. F.: Osteoid osteoma of Talus, Radiography and Clinical Photography *22:* 58, 1946.
21. Harmon, P. H.: Osteoid osteoma of mid-shaft of femur, case report, Am. J. Surg. *66:* 128 (Oct.) 1944.
22. Hatcher, C. H.: Pathogenesis of localized fibrous lesions in metaphyses of long bones, Ann. Surg. *122:* 1–16 (Dec.) 1945.
23. Henderson, M. S.: Chronic sclerosing osteitis, J.A.M.A. *82:* 945 (March 22) 1924.
24. Hitzrot, L. H.: Sclerosing osteomyelitis of carpal schaphoid, Ann. Surg. (In Transactions of N. Y. Surg. Society) *91:* 450 (March) 1930.
25. Horwitz, T.: Chronic sclerosing non-suppurative osteomyelitis of Garre; report of case with involvement of cuboid bone, Radiology *37:* 496 (Oct.) 1941.
26. Horwitz, T.: Osteoid-osteoma of astragalus, Radiology *39:* 226 (Aug.) 1942.
27. Jaffe, H. L.: Osteoid osteoma, abstract Am. J. Path. *12:* 796 (Oct.) 1936.
28. Jaffe, H. L.: Osteoid-osteoma; benign osteoblastic tumor composed of osteoid and atypical bone, Arch. Surg. *31:* 709 (Nov.) 1935.
29. Jaffe, H. L.: Osteoid-osteoma of bone, Radiology *45:* 319 (Oct.) 1945.
30. Jaffe, H. L., and Lichtenstein, L.: Osteoid-osteoma: further experience with this benign tumor of bone; with special reference to cases showing lesion in relation to shaft cortices and commonly misclassified as instances of sclerosing non-suppurative osteomyelitits or cortical bone abscess, J. Bone and Joint Surg. *22:* 645 (July) 1940.
31. Jaffe, H. L., and Mayer, L.: Osteoblastic osteoid tissue-forming tumor of a metacarpal bone, Arch. Surg. *24:* 550 (April) 1932.
32. Kleinberg, S.: Osteoid osteoma of femur; report of case, Am. J. Surg. *53:* 168 (July) 1941.
33. Kleinberg, S.: Osteoid osteoma, Am. J. Surg. *66:* 396 (Dec.) 1944.
34. Kleinberg, S.: Osteoid-osteoma; report of 5 cases, New York State J. Med. *43:* 332 1943.
35. Lapidus, P. W., and Wilson, M. J.: Osteoid osteoma; report of 3 cases, Bull. New York M. Coll. Hosp. *10:* 37, 1947.
36. Lewis, R. W.: Osteoid-osteoma; review of portions of literature and presentation of cases, Am. J. Roentgenol. *52:* 70 (July) 1944.
37. Mackenzie, W.: Painful, non-suppurative localized sclerosis of long bones; with report of 2 cases, J. Bone & Joint Surg. *29:* 49 (Jan.) 1947.
38. McKeever, F. M.: Osteoid-osteoma, West. J. Surg. *58:* 213 (May) 1950.
39. Milch, H.: Osteoid-tissue forming rumor simulating annular sequestrum, J. Bone & Joint Surg. *16:* 681 (July) 1934.
40. Miller, O. L.: Osteoid osteoma, Bull. Charlotte Mem. Hosp. *2:* 37 (Sept.) 1946.

41. Miller, J. W., and Armstrong, T. M.: Osteoid-osteoma; case report, Bull. Mason Clin. *1:* 131 (Dec.) 1947.
42. Misra, S. C., Sharma, M. L., and Singh, J.: Osteoid osteoma, Indian J. Surg. *9:* 198 (Dec.) 1947.
43. Moberg, E.: Natural course of osteoid osteoma, J. Bone & Joint Surg. *33:* 166 (Jan.) 1951.
44. Morrison, G. M., Hawes, L. E., and Sacco, J. J.: Incomplete removal of osteoid-osteoma, Am. J. Surg. *80:* 476 (Oct.) 1950.
45. Morton, H. S., and Crysler, W. E.: Osteochondritis dissecans of supratrochlear septum, J. Bone & Joint Surg. *27:* 12 (Jan.) 1945.
46. New England J. Med.: Case 27492, case records of Massachusetts General Hospital, *225:* 920 (Dec. 4) 1941.
47. New England J. Med.: Case 31432, case records of Massachusetts General Hospital, *233:* 508 (Oct. 25) 1945.
48. New England J. Med.: Case 31462, case records of Massachusetts General Hospital, *233:* 601 (Nov. 15) 1945.
49. New England J. Med.: Case 34501, case records of Massachusetts General Hospital, *239:* 931 (Dec. 9) 1948.
50. New England J. Med.: Case 35181, case records of Massachusetts General Hospital, *240:* 728 (May 5) 1949.
51. Palmer, I.: Peculair bone tumor, case report of condition described previously in literature under name osteoid-osteoma or corticalis osteoid, Acta. chir. Scandinav. *92:* 387, 1945.
52. Phemister, D. B.: Chronic fibrous osteomyelitis, Ann. Surg. *90:* 756 (Oct.) 1929.
53. Phemister, D. B.: Silent foci of localized osteomyelitis, J.A.M.A. *82:* 1311 (April 26) 1924.
54. Pines, B., Lavine, L., and Grayzel, D. M.: Osteoid osteoma: etiology and pathogenesis; report of 12 new cases, J. Internat. Coll. Surgeons *13:* 249 (March) 1950.
55. Ponseti, I., and Barta, C. K.: Osteoid osteoma, J. Bone & Joint Surg. *29:* 767 (July) 1947.
56. Pritchard, J. E., and McKay, J. W.: Osteoid osteoma, Canad. M. A. J. *58:* 567 (June) 1948.
57. Sherman, M. S.: Osteoid osteoma associated with changes in adjacent joint; report of 2 cases, J. Bone & Joint Surg. *29:* 483 (April) 1947.
58. Sherman, M. S.: Osteoid osteoma; review of literature and report of 30 cases, J. Bone and Joint Surg. *29:* 918 (Oct.) 1946.
59. Sobel, R.: Osteoid-osteoma; report of 2 cases and review of pertinent literature, Bull. Hosp. Joint Dis. *7:* 94 (Oct.) 1946.
60. Stauffer, H. M.: Osteoid-osteoma of head of radius; case report, Am. J. Roentgenol. *52:* 200 (Aug.) 1944.
61. Thoma, K. H.: Central osteomas and cementomas; diagnosis and treatment, J. Am. Dent. A. *25:* 750 (May) 1938.
62. Vaughan, W. W., Johnston, G. B., and Bugg, E. I.: Osteoid osteoma of bone. A clinical study with report of 2 cases, North Carolina M. J. *11:* 660 (Dec.) 1950.
63. Voshell, A. F., and Appleby, G. S.: Osteoid-osteoma associated with myositis ossificans; case report, Bull. School Med., Univ. Maryland, *30:* 140 (Jan.) 1946.
64. Wallace, G. T.: Some surgical aspects of osteoid osteoma, J. Bone & Joint Surg. *29:* 777 (July) 1947.
65. Weeden, W. M., and Oliva, J. J.: Osteoid osteoma, Am. J. Surg. *71:* 558 (April) 1946.
66. Wilson, H.: Extraskeletal ossifying tumors, Ann. Surg. *113:* 95 (Jan.) 1941.

TREATMENT OF RECURRING INTESTINAL OBSTRUCTION BY THE PLICATION PROCEDURE

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The development of intra-abdominal adhesions is one of the great hazards and complications of mechanical and inflammatory trauma to the visceral peritoneum. No practical maneuver is available which will prevent the formation of adhesions between damaged visceral surfaces. The tendency to form adhesions varies widely among individuals, and the ultimate determining factors which govern their formation is unknown.

Not infrequently extensive adhesions following laparotomy will develop to such a degree as to plague an individual thereafter with repeated attacks of intestinal obstruction. Numerous operations may be required to relieve the obstruction only to aggravate the situation by increased involvement. An individual may become an *abdominal invalid* suffering almost continuously from pain and varying degree of bowel obstruction accompanied by great weight loss, because eating initiates increased peristalsis and discomfort. Practically all large surgical services have patients repeatedly returning for treatment of recurrent symptoms of obstruction which eventually may require several operations to relieve the obstruction and *free* the adhesions. The simple sharp angulation of a loop of bowel of itself does not result in obstruction. Obstruction occurs most frequently at the site where a loop of bowel becomes adhered to the root of the mesentery or deep in the pelvis against its rigid walls. The sharp angulation and tension caused by the pressure of the mesentery and rigid pelvic structures seem to favor massive proliferation of adhesions. Edema at such a site readily blocks an already narrowed lumen, increases the lateral pressure due to the compression and initiates a vicious cycle. Other of the well recognized mechanisms causing obstruction are internal hernias, strangulation by bands of adhesions and volvulae between and around fixed adhesions.

Noble (1937) suggested that while intra-abdominal adhesions cannot be prevented, the site of their reformation could be controlled and subsequent bowel obstruction avoided. Loops of bowel with denudation of the serosa are sutured together to favor adhesions between adjacent segments and to obliterate pockets between adjacent leaves of the mesentery. This procedure does not allow the wall of bowel to become strongly attached at the root of the mesentery.

The Plication Procedure. The operative procedure in plication is most simple (Table 1). The mesentery of a selected loop of bowel is sutured with a continuous fine plain catgut, beginning at the base of the mesentery and proceeding outwards to the bowel. At this point the suture is tied in continuity and the stitch is con-

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tinued to appose the bowel of the two limbs to form a unit wing of the plication. It makes no difference whether the suture is placed near the mesenteric border of the bowel or near its antimesenteric surface. The limb of plicated loops will average 15 cm. in length. Succeeding segments of bowel are plicated against the preceding wings of the plication by folding the length of bowel alongside. The apices of these loops which will be the point of sharp flexion of the bowel are obviously first on one end of the aggregate loops and then on the other. This

TABLE 1

Plication procedure as applied to 47 patients

1. Extent of procedure a few inches to entire small bowel—average approximately 4 feet
2. 42 had had previous abdominal operations
3. 26 had had intestinal obstruction
4. 21 had had one to six operations for intestinal obstruction over period as long as 28 years
5. 9 had had several bouts of small bowel obstruction relieved by gastric suction
6. No operative deaths—1 death from carcinomatosis 1 year postoperatively

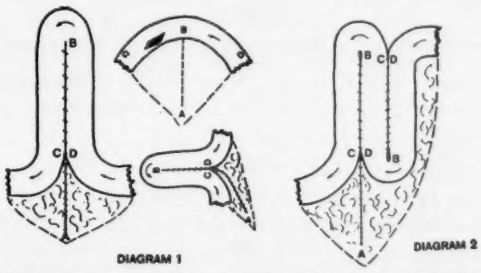

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FIG. 1. The single unit of plication is the basis for all plication. Multiple plications are simply single units added to others. In diagram 1 it will be seen that any loop or section of small bowel may be picked up and folded. As this is done the mesentery will fold, of course. A point A is taken at the end of this mesenteric folding, and this for our purpose may be taken as the surgical root of the mesentery, although it is not the anatomical base or root of the mesentery. At point A, if a suture is started, there will be no cavity left open for some other part of the bowel to enter or become caught. This point is chosen for this purpose alone, and will be called the point at the root of the mesentery with this understanding. A running suture unites lines or folds of the mesentery arbitrarily chosen from point A to the intestine, lines AC and AD in the diagram. These, when brought together, will close the mesentery completely. The suture is carried on down the intestine to the apex of the chosen wing, point B, or unit of plication. As shown, this may be closed tightly or left not quite tightly closed. Diagram 2 shows two single units combined into a three wing plication. Obviously points A are alternately on opposite sides of the mesentery. (From Noble: The Treatment of Peritonitis and Its Aftermath, 1945, A. V. Grindle, Indianapolis, Ind. publisher.)

procedure is continued to add wing after wing and plicate as much or as little of the small bowel as is indicated. The length of bowel included may vary from a few centimeters to the entire ileum and jejunum. Figure 1 shows the essential aspects of the procedure. Early in our experience we had considerable misgivings over including short segments of undamaged bowel into the plication for fear that the loop would not become adhered and would result in blind pockets and

bands as potential sites for future strangulation. The tendency for adhesions to form wherever a suture is placed in the serosa of bowel, as described by Rhoads and Singleton, Rowe and Moore assures firm union between the sutured limbs of bowel.

Freeing of Adhesions. Lysis of the adhesions is the most time consuming and tedious portion of the procedure. Most adhesions between loops of bowel can be separated easily and rapidly by blunt finger dissection with less trauma or likelihood of opening into the bowel than by sharp dissection.

The heavy adhesions, especially those to the root of the mesentery and rigid structures of the abdomen and pelvis, will require sharp dissection.

Effective use of finger dissection shortens the procedure considerably. Freeing the entire length of small bowel requires an average time of 30 minutes. If the freed loops of bowel are returned to the abdominal cavity immediately, the patient withstands the procedure well. Subsequently, the bowel is delivered into the wound as it is plicated, and the plicated loops returned to the abdominal cavity or kept well covered.

TABLE 2

Age distribution of 47 patients subjected to plication operation

Age	No. of patients
0–10	6
10–20	3
20–30	4
30–40	12
40–50	15
50–60	2
60–70	5

The Choice of Patients for the Plication Procedure. The procedure of plication was first used by the authors to fix the distal portion of the ileum to the ascending colon to prevent repeated intussusception. The patient, a 2 year old Negro child, had been operated upon about eight months previously for an earlier ileo-ileocecal intussusception. Following the attachment of the distal end of the ileum along the ascending portion of the cecum and ascending colon, no further intussusception has occurred. Approximately 40 cm. of distal ileum had been involved in the intussusception and the serosa was sufficiently damaged to favor the formation of adhesions and were included in the plication.

Initially, only those patients with severely damaged serosa of the small bowel were selected for plication, and then segments usually not exceeding 3 to 4 feet were included. The favorable results and our increased experience with the procedure has encouraged the inclusion of all portions of small bowel with denuded surfaces. The uncertainty as to the nature of the adhesions buried in large masses of omentum and bowel has directed that practically all adhesions be lysed if the patient's condition permits. All traumatized bowel is included in the subsequent plication. It is unnecessary to separate two segments of bowel which are

included in a simple side to side adhesion. It must be ascertained, however, that there is no opening along the root of the mesentery between these loops. If such an opening exists, it is closed. Frequently, a segment of normal bowel is interposed between segments of traumatized bowel. The two traumatized areas are plicated individually, and the normal intravening segment of bowel is left undisturbed.

Plicated masses of small bowel would seem likely to rotate about the narrow root of the mesentery immediately postoperatively and result in massive strangulation. This complication has not occurred in this series. As a precaution against such an eventuality, the plicated masses have at times been sutured with interrupted catgut to more or less fixed structures such as the transverse mesacolon. Within a very short time these masses of plicated bowel become fixed to adjoining structures thereby preventing future rotation.

Preoperative Preparation of the Patient. Varying degrees of small bowel obstruction modify the preoperative preparation. Gastric suction is instituted if signs and symptoms of strangulation are absent, and an attempt is made to decompress the abdomen. Ideally, it is hoped to return the patient preoperatively to a low residue diet and administer neomycin-sulfathalidine combination to eliminate the intestinal bacteria. The intestinal antiseptics are used as a precaution against soiling following the inadvertent opening of the bowel. Not infrequently these patients are completely obstructed and do not resolve under conservative gastric suction. They may present signs and symptoms of strangulation. Immediate exploration is then undertaken without special preoperative preparation, except rapid correction of dehydration, electrolyte, fluid balance, and anemia. During an emergency operation, the distended bowel may accidentally be opened. In this event, the opened segment is transferred to a sterile wash basin and quarantined from the remainder of the operative field by sterile sheets. The short length of bowel including the vent extends between the folds of the sheet into the wash basin. One member of the surgical team handles the bowel in the contaminated area. The others manipulate the distended bowel to evacuate the entrapped liquid and gas with a minimum of trauma. At the termination of this maneuver the contaminated portion of bowel is sponged off with saline solution, dried, then covered with a pack moistened with a 1 per cent neomycin solution and returned to the sterile field. Appropriate occluding clamps are immediately applied and the entire small bowel flooded with 1 per cent neomycin injected through a small needle into the lumen. As much as 1000 cc. of 1 per cent neomycin solution may be used. It has been repeatedly demonstrated that within 30 minutes a specimen aspirated from the bowel contains no viable bacteria. If the defect in the bowel is too large to permit direct closure, it is effected by the procedure demonstrated in figures 2 and 3.

Should necrotic bowel require resection, the bowel is clamped within the area which will be sacrificed ultimately and the damaged segment rapidly resected. Via the distal end, the dilated proximal segment is decompressed as previously described and the small bowel flooded with 1 per cent neomycin. The site for reanastomosis is selected, prepared for resection, clamped with beveled clamps and

the additional tissue removed. The anastomosis is done using the technic illustrated in figures 4 and 5 unless edema of the viscus precludes bowel suture. The anastomosis is incorporated in the plication which completely covers the line of suture.

Postoperatively, these patients are placed on gastric suction. If there has been no bowel suture, the suction is discontinued as soon as peristalsis is re-established

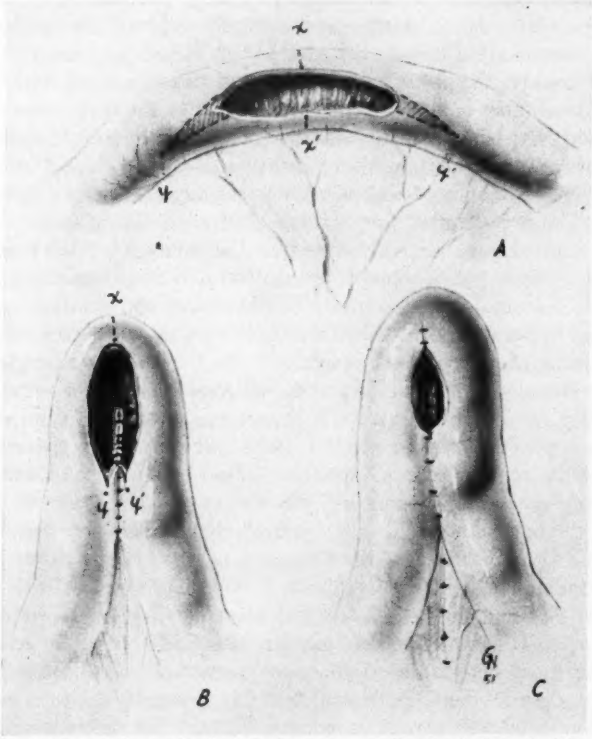

FIG. 2. Illustration of the method of closing a long defect in the bowel without resection. The defect may be enlarged, as indicated in A, when it is necessary to obtain a sufficiently long stoma. B shows the two posterior rows of sutures in place. The bowel has been folded on itself around points x x. C shows the incomplete anterior suture. The choice of suture is left to the individual surgeon. Note that a locked stitch should not be used to suture bowel, because it results in pressure necrosis under each locked loop of the suture. (From Poth: A Method of Closing Defects in Bowel, S. G. & O., *93:* 606–608, 1951.)

and a low residue diet allowed. Following anastomosis or simple closure of bowel, the gastric suction is continued until the patient passes flatus or has a bowel movement. The feces of the first bowel movement are sterile as a result of the neomycin introduced during the operation. Should conditions warrant, the patient is placed on neomycin-sulfathalidine mixture postoperatively for a period of 9 to 12 days for continued suppression of the intestinal bacterial flora. No special

procedures are instituted to increase peristalsis immediately postoperatively. A small percentage of patients will show mild signs and symptoms of partial in-

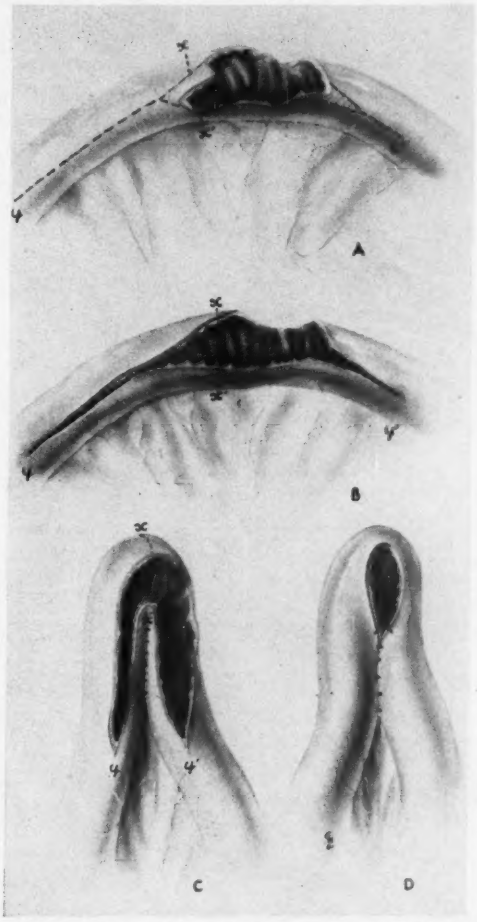

FIG. 3. An extensive, ragged defect involving more than half the diameter of the bowel. The traumatized edges of the defect are trimmed as indicated in A and B. The bowel will be folded or plicated on itself around points x x. It will be noticed that a long incision is made to point y. The points x x are selected so as to give the full diameter of one limb of the bowel for filling in the defect at its greatest extent. C shows the approximation of points y and y and the placement of the outer posterior row of interrupted sutures. The inner posterior row is placed before beginning the anterior closure. D shows the incomplete anterior row of sutures. (From Poth: A Method of Closing Defects in Bowel. S. G. & O., *93:* 606–608, 1951.)

testinal obstruction for a period varying from a few days to three weeks. These symptoms then disappear. In no instance has it been necessary to reoperate upon

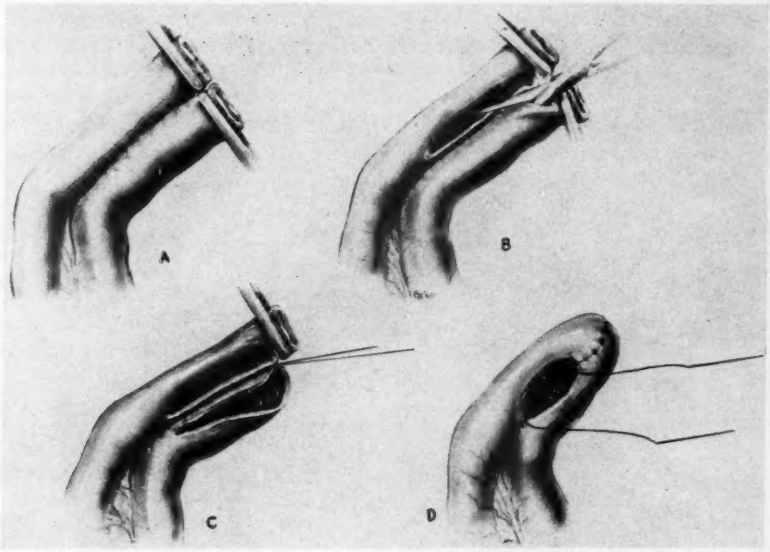

FIG. 4. Illustration of the technic applied to a blind double-ended anastomosis. (From Poth: A Technique for Suturing Bowel. S. G. &. O., *91:* 656–659, 1950.)

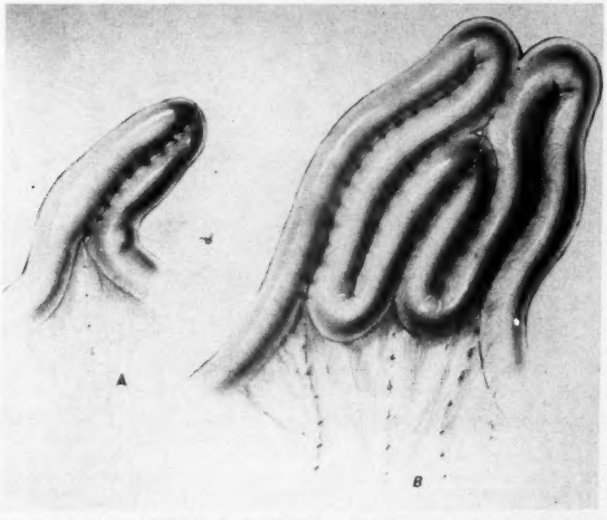

FIG. 5. A demonstration of the technic illustrated in figure 4 being applied to anastomosis of the small bowel coupled with plication of damaged loops of bowel to control the formation of adhesions and reduce the hazard of intestinal obstruction. Following completion of an anastomosis as illustrated in figure 4, adjacent loops of bowel are plicated. The end of the anastomosis is used as the apex and starting point for the plication. (From Poth: A Technique for Suturing Bowel, S. G. &. O. *91:* 656–659, 1950.)

any of these patients because of obstruction in the plicated portion of the bowel. One patient required re-exploration because of evidence of continued partial obstruction several months after the initial procedure, but the recurring obstruction was in a portion of bowel not incorporated in the initial plication.

Use of the plication procedure has removed our former dread of operating upon patients with many intra-abdominal adhesions. During our earlier experience with this operation, we took down only those adhesions which apparently were directly involved in obstruction. Adhesions not causing obstruction were not disturbed even though it was believed that these might be sites of future difficulty. Now all adhesions are lysed. It cannot be accurately determined whether or not, hidden deep in a mass of adhesions, an area might not be sufficiently involved to cause future trouble.

The almost complete freedom of postoperative complications is a welcome surprise. Postoperative difficulties with recurrence of a fairly high percentage of obstructions requiring reoperation was feared. This has not been our experience.

TABLE 3

Postoperative complications occurring in 47 patients following the plication procedure for the relief of adhesions

1. Wound disruptions—2
2. Postoperative hernia—1
3. 3 cases of recurrent obstruction
   1—carcinomatosis—terminal
   1—partial obstruction—descending colon—carcinoma
   1—recurrence of chronic volvulus not involving plicated bowel
4. Thrombophlebitis—1
5. About 30% of patients have abdominal cramps occasionally as long as three weeks postoperatively

The rather hopeless attitude towards the so-called *abdominal invadlid* is changed into one of confidence since we are able to remedy this difficulty. Credit for this procedure should go to Dr. Thomas B. Noble, Jr., who described the procedure in 1937 and wrote a monograph on this technic in 1945. Our interest in this method of management of extensive intra-abdominal adhesions follows the description of the method by Lord, Howse and Jolliffe in 1949.

The greatest postoperative difficulty is the recovery from morphine addiction acquired during the preceding years of abdominal discomfort. It is significant that in this series of practically 50 patients, subjected to the procedure, not a single instance of obstruction has occurred subsequently in the area plicated.

DISCUSSION

It is recommended that boldness be exercised in the application of this procedure. No damaged bowel should be replaced into the peritoneal cavity without being incorporated in a plication. It must be anticipated that there will be compli-

cations and, at times, failures but the benefit and gain is so great that the use of this procedure must be given our full endorsement.

CONCLUSIONS

1. The Noble Plication Procedure is an excellent method of management of recurring attacks of partial or complete obstruction due to intra-abdominal adhesions.

2. The conversion of *uncontrolled adhesions* to *controlled adhesions* gives an excellent solution to a most troublesome complication which, in the past, has been looked upon as quite hopeless by the profession.

REFERENCES

1. Lord, J. W., Jr., Howse, E. L., and Jolliffe, N.: Surgical management of chronic recurrent intestinal obstruction due to adhesions, Ann. Surg. *129:* 315 (March) 1949.
2. Noble, T. B., Jr.: Plication of small intestine as prophylaxis against adhesions, Am. J. Surg. *35:* 41 (Jan.) 1937.
3. Noble, T. B.: Monograph, Treatment of Peritonitis and Its Aftermath, Indianapolis, Indiana, A. V. Grindle, 1945.
4. Rhoads, J. E., in discussion of paper by Robbins, G. F., Brunschwig, A., and Foote, F. W., Jr.: Deperitonealization: clinical and experimental observations, Ann. Surg. *130:* 466 (Sept.) 1949.
5. Singleton, A. O., Rowe, E. B., and Moore, R. M.: Failure of reperitonealization to prevent abdominal adhesions in the dog, American Surgeon *10:* 789 (Aug.) 1952.

THE TECHNIC OF ADEQUATE COMMON DUCT EXPLORATION USING A NEW TYPE FLEXIBLE PROBE AND DILATOR

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Complicated gallbladder and common duct operations try the patience and ingenuity of the most accomplished surgeons. Cholangiograms taken during operation have helped in the reduction of poor results but of greater importance has been the emphasis placed upon adequate exposure, the recognition of anatomical anomalies, and gentle, thorough exploration of the ducts, using the transduodenal approach when necessary.

There is no group of symptoms or signs, either prior to or at the time of operation, that will definitely rule in or rule out the presence of common duct stones. As a result of this fact, common bile duct exploration is being carried out in an increasing number of patients. Dissatisfaction with available instruments for exploration of the ducts led to the development of the flexible probes and dilators described (fig. 1).*

Before undertaking common duct exploration, which prolongs the time of operation, and unless very carefully done, increases the morbidity and mortality, the surgeon must have clearly in mind what he expects to accomplish by common duct exploration and what steps he is going to carry out to insure an adequate exploration without producing undue trauma to the biliary system.

The indications for common duct exploration have become fairly well standardized and, assuming competence of the surgeon, may be divided into:

A. Conditions making exploration mandatory (absolute).
   1. Palpation of stones in the ducts.
   2. Clinical jaundice or a history of recent or repeated attacks of jaundice.
   3. A chronically infected contracted gallbladder containing small stones.
   4. A dilated or thickened common duct.
   5. Sediment in bile aspirated from the common duct.
   6. A gallbladder containing small stones with a wide patent cystic duct.

B. Conditions which may or may not require exploration (relative).
   1. Cholelithiasis and laboratory evidence of sub-clinical jaundice. (A serum bilirubin of 0.5 to 0.7 mg. per 100 cc.)
   2. A non-calculous gallbladder with a good history of biliary colic.
   3. A normal gallbladder with edema or induration in the head of the pancreas.
   4. Inability to empty a dilated gallbladder with moderate digital pressure. (May indicate a stone impacted in the ampulla or a tumor of the papilla.)

From the Department of Surgery, University of Utah Medical School. Presented during the annual assembly of The Southwestern Surgical Congress, October 20-22, 1952, Dallas, Texas.

* Made by Surgical Supply Company, Salt Lake City, Utah.

Technic:

If operative cholangiograms are to be of any value the patient must be placed on a table equipped with a Bucky grid and some type of plate changer. If preparations for cholangiography are not made prior to surgery the increased operating time and trauma of the procedure far outweigh the value of any information to be gained.

FIG. 1. Flexible gall duct probes and dilators. These probes will produce less trauma than rigid probes.

Anesthesia must be adequate and the incision large enough to provide good exposure. For difficult cases the right rectus incision seems to be more satisfactory. It allows a more complete general abdominal exploration.

If the gallbladder is distended it should be partially emptied using a syringe and a no. 17 gage needle. Traction sutures are then placed in the lower third of the gallbladder and in the forward bulging portion between the body and the cystic duct (ampulla). When these are tightened the hepato duodenal ligament is made taut and can safely be incised, thereby exposing the cystic duct. This incision is then carried down to visualize the junction of the cystic and common ducts and the proximal 2 cm. of the common duct.

The operating surgeon should then go to the left side of the table and, using

his right hand he should carefully palpate the ducts and the head of the pancreas. If no stones are palpated aspiration of the common duct is done with a 10 cc. syringe and a no. 22 gage needle. The character of the aspirated bile is noted.

If any of the positive indications are present surgical exploration of the common duct is done. In an average risk patient exploration is done for any of the relative indications, but in a poor risk patient cholangiography is done and if the cholangiogram appears to be normal a decision to forego exploration may be made. Preoperative determinations of the prothrombin time, the serum protein

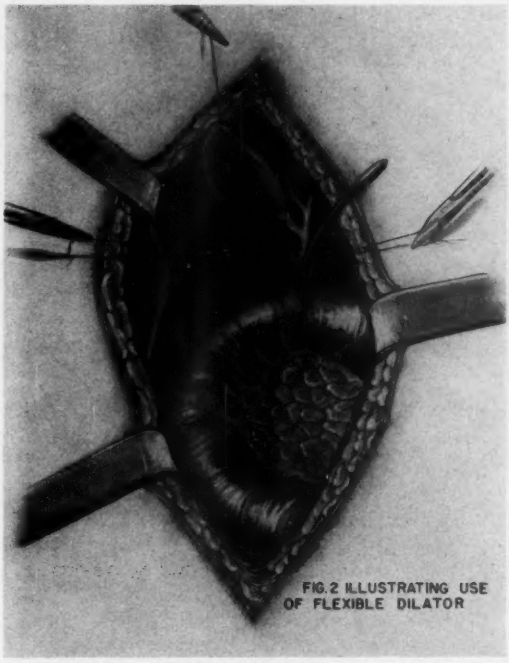

FIG. 2. Flexible probe and dilator in the common bile duct extending into the duodenum

and blood volume serve as guides to the extent of the operative procedure that can be safely performed.

Surgical exploration of the duct is carried out by placing two no. 000 intestinal chromic catgut sutures, for traction, in the common duct ½ to 1 cm. distal to the junction of the common and cystic ducts, and making a longitudinal incision 5 to 7 mm. long between the sutures, using a long handled knife and a no. 12 blade. Escaping bile is aspirated and the opening in the duct is carefully observed as stones may be washed out by the bile. A 3 mm. flexible dilator is then inserted into the common duct and, if possible, passed without force through the sphincter of Oddi into the duodenum (fig. 2). Palpation of the duct over the dilator is then carefully done, and if any stones are palpated they are removed.

The sphincter is then enlarged by passing the graduated dilators to the 7 mm. size. This degree of dilatation can be done safely and is adequate to facilitate the smaller stones to pass into the duodenum. The operating surgeon then returns to the right side of the table and the 3 mm. flexible dilator is passed into the hepatic ducts and the ducts are palpated over the dilator. After removal of the dilator, thorough, gentle saline irrigation of the ducts above and below the incision is done through a catheter. Using the technic of duct drainage described by Allen, a no. 10 F. catheter is fixed in the duct with interrupted no. 000 chromic catgut sutures (fig. 3). A catheter is preferred to a T tube as it can be quickly placed

FIG. 3. Method of placing a catheter in the common bile duct and holding it in place with a fine catgut suture.

without trauma but if a severe cholangitis with marked dilatation of the ducts is present a T tube should be used, since prolonged drainage will be necessary. Patency of the sphincter is then tested by injection of normal saline through the catheter or T tube without force. It should enter and distend the duodenum without leaking. If distension of the duodenum does not occur following the injection of the saline it may be assumed that there is an obstruction of the papilla which the probing has failed to detect. It is possible to apparently feel the tip of the dilator in the duodenum when it is actually the papilla which has been forced ahead of the dilator against the anterior duodenal wall, the dilator never having passed through the sphincter. If this is suspected a cholangiogram should

be taken. In the absence of positive evidence of patency of the sphincter transduodenal exploration is mandatory.

The first and most important step in transduodenal exploration is mobilization of the descending portion of the duodenum by incising the peritoneum laterally and reflecting the duodenum medially and anteriorly. Exposure of the papilla can then be easily accomplished through a small longitudinal incision in the anterolateral wall of the duodenum. If a stone is impacted in the ampulla, the papilla may be carefully dilated and the stone removed. Simple transverse closure of the duodenum is then made.

If a stricture of the duodenal portion of the duct is present it should be incised and dilated and the catheter previously placed in the common duct should be removed. The 3 mm. flexible dilator should be passed up through the papilla and out through the common duct incision. The long limb of a Cattell type T tube is tied over the tip of the dilator and drawn down through the common duct into the duodenum. The common duct is then closed around the Cattell tube. The incision in the duodenum is sutured transversely and the duodenum restored to its former position.

DISCUSSION

With the technic outlined using careful surgical exploration with gentle dilatation of the sphincter of Oddi and operative cholangiograms as complementary procedures poor results will be reduced to a minimum. To rely entirely upon abnormal operative cholangiograms as the criterion for surgical exploration of the ducts will result in frequent errors.[1] The judicious use of operative cholangiograms will lessen the morbidity and mortality in poor risk patients who do not have absolute indications for choledochostomy. Gentle moderate dilatation of the sphinceter of Oddi using graduated olive tipped Bakes dilators has been widely accepted.[2, 3, 4] The use of the flexible graduated dilators described here greatly facilitates the ease of the procedure, reduces the time required and minimizes trauma.

SUMMARY

The absolute and relative indications for surgical exploration of the common bile duct have been listed.

A technic for adequate exploration of the ducts using a new flexible probe and dilator has been presented.

The importance of operative cholangiograms in poor risk patients without absolute indications for surgical exploration of the common duct and for assuring patency of the papilla of Vater during surgical exploration of the duct has been discussed.

REFERENCES

1. Allen, A. W.: Diagnosis and treatment of stones in the common bile duct, Surg., Gynec. & Obst. *62:* 347 (Feb. No. 2A) 1936.
2. Bakes, J.: Sur drainagelosen Gallencherurgin und der Methodischen Dilatation der Papille, Zentrabl f. Chir. *55:* 1858 (July 28) 1928.
3. Cattell, R. B.: Use of a long T-tube in surgery of biliary tract, S. Clin. North America *28:* 659 (June) 1948.
4. Mixter, C. G., Hermanson, L., and Segal, A. L.: Operative cholangiography, an evaluation of 406 cases, Ann. Surg. *134:* 346 (Sept.) 1951.

ANNULAR PANCREAS

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Annular pancreas is a rare anomaly. Only 31 cases treated surgically have been reported previously in the medical literature and approximately 40 additional cases have been found at autopsy and in anatomical dissections. One additional case operated upon is herewith reported. It is of interest that of these 32 cases, 24 have been reported since 1940. This fact would strongly suggest that the condition is more common than it has been supposed to be and that it is being discovered more frequently in recent years due to increased alertness on the part of the profession, especially by roentgenologists and surgeons. That a correct preoperative diagnosis may not be made is not surprising since meticulous roentgenologic technic and careful and generous exposure by the surgeon at the time of operation may be required to identify the lesion.

The purpose of this paper is to call to the attention of the profession this condition, to review briefly the reported literature on the subject and to report a case with several interesting features. The subject has been ably reviewed recently by numerous writers, especially Silvis, Payne, Warren and others.

Etiology. The lesion is a congenital one and according to Ravitch and Woods is the only congenital lesion of the gastrointestinal tract which in most cases produces no symptoms until late in life. Warren believes that it is formed by faulty fusion of the ventral and dorsal anlage of the primitive pancreas and that it is probably influenced by disturbances in the rotation of the intestine. Payne believes that it is due to failure of the ventral anlage of the pancreas to rotate with the duodenum. The lesion consists of a band or ring of pancreatic tissue which encircles the duodenum in its second portion. This ring has its own duct system. It is said that the ring may be incomplete, in which case the defect is anterior. This encirclement produces obstruction of the duodenum of a greater or lesser degree. The condition has been reported in all age groups including the newborn and the series operated upon includes patients ranging in age from 3 days to 74 years. Twenty-one of the 32 cases operated upon were males and 11 were females. Other congenital anomalies have been reported frequently. According to Gross and Chisholm 20 per cent of the cases found in autopsy and anatomical department specimens have other congenital abnormalities such as atresias of the duodenum or anus and cardiac defects.

Pathology. Grossly and microscopically the involved portion of the pancreas, as well as the rest of the pancreas, may be entirely normal. In several reported cases, however, pancreatitis has been found on biopsy. According to Lehman, of

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TABLE I

Tabulation of all surgically treated cases of annular pancreas

Case No.	Reported by	Year	Age	Sex	Operation	Result	Comment
1	Vidal	1905	3 days	M	Posterior gastroenterostomy	Cured	Also congenital atresia of duodenum
2	dos Santos	1906	26 yrs.	F	Posterior gastroenterostomy	Died	Chronic pancreatitis. Diagnosis postmortem
3	Lerat	1908	46	F	Resection of ring	Cured	Drainage for 13 days
4	Smetana	1928	74	M	Posterior gastroenterostomy	Died	Fibrosis of pancreas. Diagnosis postmortem cancer of cystic duct
5	Howard	1930	46	F	Division of ring	Cured	Pancreatic fistula requiring second operation
6	Brines	1930	35	M	Drainage of pancreas	Died	Diagnosis postmortem
7	Zech	1931	27	F	Duodenoplasty	Cured	Small fistula. Duodenum did not reexpand postoperatively
8	Brines	1931	44	M	Posterior gastroenterostomy	Died	Diagnosis postmortem
9	Truelsen	1940	35	M	Posterior gastroenterostomy duodenoplasty	Cured	
10	Lehman	1942	23	M	Partial resection of ring	Recovery but persistent symptoms	Persistence of deformity by x-ray postoperatively
11	Gross & Chisholm	1944	3 days	F	Duodenojejunostomy	Cured	
12	Custer & Waugh	1944	74 yrs.	M	Gastric resection	Cured	Also benign gastric ulcer
13	Goldyne & Carlson	1946	26	M	Partial resection of ring	Cured	Persistence of gastric & duodenal residue by postoperative x-ray
14	Nedelec	1946	17	M	Posterior gastroenterostomy	Cured	
15	Ohlmacher & Marshall	1946	27	M	Gastric resection	Cured	

TABLE I—*Cont.*

Case No.	Reported by	Year	Age	Sex	Operation	Result	Comment
16	Brown, Bingham & Cronk	1948	53	F	Division of ring	Died	Subdiaphragmatic abscess, duodenal fistula
17	Burger & Alrich	1949	4 days	F	Partial resection of ring. Duodenoplasty	Died	
18	Baker & Wilhelm	1950	59 yrs.	M	Gastric resection	Cured	Also benign gastric ulcer
19	Ravitch & Woods	1950	57	M	Partial resection of ring. Duodenojejunostomy 37 days later	Cured	
20	Ravitch & Woods	1950	3 days	F	Duodenojejunostomy	Cured	
21	Ravitch & Woods	1950	8	M	Gastroduodenostomy	Cured	Malrotation of intestine. Complete duodenol atresia
22	Archer—Reported 1950 by Haden		3½ yrs.	M	Partial resection of ring	Recovered	Persistence of partial duodenal obstruction
23	Conroy & Woelfel	1951	30	M	Division of ring	Cured	
24	Grotjan. Reported by Conroy & Woelfel	1939	26	F	Gastroenterostomy	Cured	
25	Payne	1951	33	M	Partial resection of ring	Recovered	Persistence of duodenal obstruction
26	Cattell	1951	Adult	M	Division of ring. Gastric resection	Cured	Postoperative jejunal fistula. Roux exclusion & resection of fistula
27	Silvis	1951	37	M	Partial resection of ring	Cured	Pancreatic fistula
28	Bickford & Warren	1951	25	M	Posterior gastroenterostomy	Cured	
29	Warren	1952	37	M	Gastric resection & excision of anterior part part of ring.	Cured	Pancreatic & jejunal fistula. Partial wound disruption. Had duodenal ulcer

Case No.	Reported by	Year	Age	Sex	Operation	Result	Comment
30	Warren	1952	22	F	Cholecystectomy & choledochostomy	Diarrhea & cramps	Had previous division of stomach antrum & gastroenterostomy
31	Warren	1952	41	F	Gastroenterostomy. Vagotomy. Choledochostomy	Cured	Postoperative diarrhea for 10 days
32	Castleton, Morris & Kukral	1952	41	M	Posterior gastroenterostomy Choledochostomy	Cured	Concomitant amebiasis, chronic ulcerative colitis & severe pancreatitis

10 cases which have been operated upon and tissue removed for a biopsy, microscopic examination showed 5 with chronic interstitial pancreatitis. In Bickford's case the pancreas was so hard that it was thought to be malignant. No tissue for biopsy was taken but it seems certain that chronic pancreatitis was present in view of the good results. Our own case showed an acute and subacute pancreatitis in the biopsy of the annular portion and a very severe chronic pancreatitis of the body of the pancreas. It would appear that the high incidence of pancreatitis in the reported cases may be significant despite the small number of cases reported. The possible relationship of pancreatitis to annular pancreas is not clear but it has occurred to us that it is probably more than a casual one. In our case it would appear that, although the annular arrangement had undoubtedly been present since birth, the signs and symptoms of obstruction might have been brought on by the development of acute pancreatitis with swelling of the gland and that this swelling tended to compress the duodenum to the point where clinical signs became manifest.

Obstruction of the duodenum at the site of the lesion with dilatation above is the most common pathologic finding, the degree of obstruction varying considerably. Compression of the common bile duct may also occur. Peptic ulcer of the duodenum or stomach has also been found in several cases. In fact, here again, the incidence of associated peptic ulcer would seem to be higher than is normally found. In our case there was an associated chronic ulcerative colitis and amebiasis. These associated conditions were apparently entirely coincidental since they have not been reported in any of the previous cases. We have considered the possibility that this patient's diarrhea might have been due to pancreatic insufficiency in view of the severity of the pancreatitis, but we have found nothing to indicate such a relationship. The stools were watery and bloody and not fatty and bulky, as they are in the case of steatorrhea due to pancreatic insufficiency.

Signs and symptoms. The clinical symptoms are those of upper gastrointestinal tract obstruction with epigastric pain, anorexia, nausea and vomiting. The intensity of the symptoms vary directly with the degree of obstruction. The lesion may exist throughout life without symptoms of any kind, or symptoms may develop at any age whenever obstruction occurs. The symptoms may be mistaken for peptic ulcer, and as noted above, ulcer frequently coexists and may lead to massive gastrointestinal hemorrhage. A correct preoperative diagnosis has been made only once previously although it was suggested as a possibility in our case by one of us. In the newborn, jaundice may be present and if untreated may result fatally within a few months.

The outstanding sign of this condition is the finding by roentgenography of a constriction of the second portion of the duodenum. This is described as a narrowing by a ring-like constriction of the second portion of the duodenum. In our case the roentgenogram report by Dr. Ralph Meyer is as follows: "There is a constricting lesion, in the second portion of the duodenum. We believe roentgenographic evidence favors an inflammatory process, or an extrinsic neoplasm, rather than a primary neoplastic lesion, although the possibility of a primary neoplastic lesion cannot be excluded."

Diagnosis. The diagnosis is usually not made because this condition is not thought of by the examiner. It may be made by (1) signs of upper gastrointestinal obstruction, (2) roentgenographic findings of a band-like constriction of the second portion of the duodenum with dilatation of the duodenum above and delay in the progress of barium past the constricted area.

Treatment. The treatment is necessarily surgical. Several types of procedures have been tried including incision of the band, excision of the constricting band, gastric resection, gastroduodenostomy, gastrojejunostomy, plastic procedure on the duodenum and combinations of these procedures. Direct attack on the obstructive lesion by excision of the annular portion of the pancreas would appear to be the most logical approach. However, this procedure has not proved satisfactory in several instances. In some cases where this has been done, pancreatic fistulas have developed, one of them to such a degree that reoperation was necessary. Gross and Chisholm state that, even after resection of the ring, duodenal obstruction may not be relieved because of extrinsic scar tissue in the duodenal wall. Gastric resection would seem to be indicated if there is an associated peptic ulcer but in the absence of ulcer some type of by-passing procedure, especially gastrojejunostomy appears to be the operation of choice. Whether gastrojejunal ulcer will develop after such a procedure is not known, but so far none has been noted in any of the reported cases. In our case a posterior gastrojejunostomy was done followed by choledochostomy. The latter was done to provide prolonged drainage of the common duct with the hope of relieving the chronic pancreatitis. A T tube was placed in the common duct and was removed at the end of 9 months. In 2 of Warren's cases choledochostomy was done in addition to either gastric resection or gastroenterostomy. In both cases the common duct was opened and drained because of dilatation of the duct. He also did a vagotomy in addition to gastroenterostomy and choledochostomy in 1 case. All 3 of his cases had a history of associated duodenal ulcer.

CASE REPORT

A 41 year old white male steel worker had always been well until about September 1945 when he developed severe diarrhea. He had 8 to 15 stools per day, and was treated elsewhere conservatively with chemotherapy and responded well. He was admitted to a hospital in September and November 1950 and in April 1951, with similar complaints with the addition of blood in the stools and abdominal cramps. There was an 8 pound weight loss. Each time he was treated with antibiotics and cortisone, and each time sigmoidoscopy revealed evidence of nonspecific ulcerative colitis. His last admission in April 1951 revealed Endamoeba histolytica trophozoite and stool culture was positive for Proteus morganii. On his last admission, the patient again was treated with intensive antibiotics, sulfa drugs and antiamebic therapy, (emetine and diodoquin) on which he improved.

On the fourth hospital admission May 22, 1951 the patient was found to be improved, and he was returning merely for further antiamebic therapy. Proctoscopy at that time

FIG. 1

FIG. 2

FIG. 1. Roentgenogram taken 1 year before admission to hospital showing duodenal defect and gastric retention.

FIG. 2. Roentgenogram showing duodenal defect and gastric retention a few days before operation.

also showed improvement. In October 1951 he had a sudden onset of severe bloody diarrhea and abdominal cramps which was believed by his physician to be acute infectious enteritis and/or recurrence of the amebic colitis. He was treated with gantrisin and within 24 hours his diarrhea subsided. Following this episode, the patient had recurrent nausea and vomiting and epigastric pain radiating occasionally to the back. The pain was constant in character with exacerbations. Physical examination at that time was negative, except for localized tenderness in the right upper quadrant. Laboratory examinations revealed negative serologic reactions of the blood. The urine showed a very slight trace of albumin, a few hyaline casts, but was otherwise negative. The hemogram was normal. The blood urea nitrogen was 24 mg. per 100 cc. The fasting blood sugar was normal. The serum amylase was 255 mg. per 100 cc. Roentgenogram of the abdomen did not reveal any lesion. The gallbladder series showed a normal functioning gallbladder without stones. Roentgenographic examination of the stomach and duodenum was reported as follows: "there is a constricting lesion in the second portion of the duodenum. We believe roentgenographic evidence favors an inflammatory process or an extrinsic neoplasm, rather than a primary neoplastic lesion, although the possibility of a primary neoplastic lesion cannot be excluded."

A surgical consultation was held and exploratory laparotomy was recommended and carried out on Dec. 3, 1951. Although the stomach and gallbladder were normal they were adhered to the transverse colon and mesocolon. There was a constricting ring of pancreas ½ to ⅔ of an inch in diameter surrounding the second part of the duodenum. A biopsy

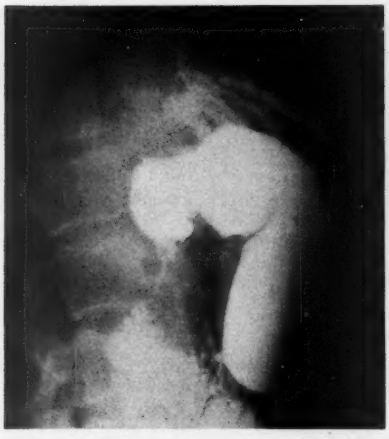

FIG. 3

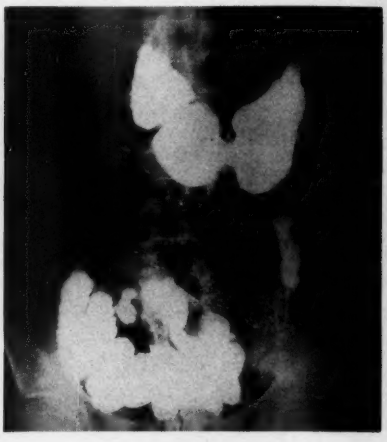

FIG. 4

FIG. 3. Lateral roentgenogram showing constriction of the duodenum
FIG. 4. Roentgenogram showing 6 hour retention of barium

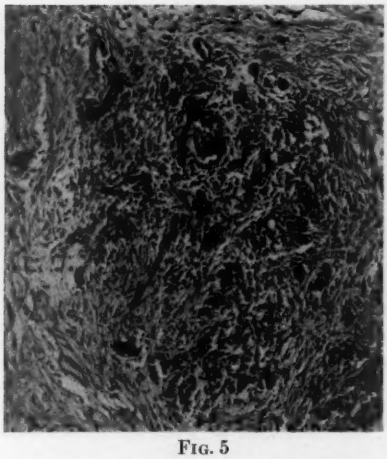

FIG. 5

FIG. 6

FIG. 5. Low power view of the pancreas showing severe chronic pancreatitis
FIG. 6. High power view showing severe pancreatitis

specimen of the pancreas was taken and a frozen section revealed a benign lesion. The pancreas felt hard, nodular and enlarged. A posterior gastroenterostomy was done followed by choledochostomy with drainage of the common duct with a T-tube.

The patient was last admitted Jan. 25, 1952 for treatment of exacerbations of ulcerative

amebic colitis and/or pancreatitis. He was given conservative therapy of banthine, bland diet and gantrisin. His condition improved and he was discharged. He was seen in early September. He was working regularly. His weight had increased from 112 pounds at the time of hospital discharge to 152 pounds. There was no evidence of ulcerative amebic colitis and he felt well.

SUMMARY AND CONCLUSIONS

Annular pancreas is a rare anomaly but it is being reported with increasing frequency in recent years probably due to increased alertness on the part of the profession. It rarely has been diagnosed preoperatively. Even at operation a diagnosis is not easily made. Although congenital in origin an annular pancreas often does not become manifest until later in life when it produces clinical and roentgenographic evidence of obstruction in the second part of the duodenum. It is frequently associated with peptic ulcer and chronic interstitial pancreatitis as indicated in the reported cases. Its treatment is surgical. At present it appears that a short circuiting procedure is the method of choice except when it is associated with peptic ulcer in which case gastric resection may be indicated.

We are reporting 1 case of this condition. It was unusual in that it was associated with amebiasis and chronic ulcerative colitis as well as with a very severe form of chronic pancreatitis which involved not only the annular portion of the pancreas, but the body as well. This is the thirty-second surgical case reported. This case was treated by prolonged choledochostomy because of the chronic pancreatitis, and by posterior gastrojejunostomy with excellent results.

REFERENCES

1. Baker, J. W., and Wilhelm, M. C.: Annular pancreas; report of surgical case with a 2 year followup, Gastroenterology *15:* 545 (July) 1950.
2. Baldwin, W. M.: A specimen of annular pancreas, Anat. Rec. *4:* 299 (Aug.) 1910.
3. Bickford, B. J., and Williamson, J. F. C. L.: Annular pancreas, Brit. J. Surg. *39:* 49 (July) 1951.
4. Brines, O. A.: Annular pancreas, involved in acute hemorrhagic pancreatitis, Ann. Surg. *92:* 241 (Aug.) 1930.
5. Brines, O. A.: Annular pancreas associated with peptic ulcer, Am. J. Surg. *12:* 483 (June) 1931.
6. Brown, M., Bingham, D. L. C., and Cronk, L. B.: Annular pancreas; description of case, Gastroenterology *11:* 367 (Sept.) 1948.
7. Burger, R. E., and Alrich, E. A.: Annular pancreas, South. Surgeon *15:* 85 (Feb.) 1949.
8. Cattell, R. B.: Discussion of article by R. L. Payne, Annular pancreas, Ann. Surg. *133:* 762 (June) 1951.
9. Chapman, J. L., and Mossman, H. W.: Annular pancreas accompanied by aberrant pancreatic nodule in duodenum, Am. J. Surg. *60:* 286 (May) 1943.
10. Conroy, C. F., and Woelfel, G. F.: Annular pancreas, Surgery *29:* 902 (June) 1951.
11. Cunningham, G. J.: Annular pancreas, Brit. J. Surg. *27:* 678 (April) 1940.
12. Custer, M. D., and Waugh, J. M.: Ann. pancreas with secondary dilatation of duodenum; report of case, Proc. Staff Meet., Mayo Clin. *19:* 388 (July) 1944.
13. dos Santos, R.: Deux Lesions Rare du Duodenum, XV Congress International de Med. Lisbon *9:* 419, 1906.
14. Goldyne, A. J., and Carlson, E.: Annular pancreas causing duodenal obstruction, Am. J. Surg. *71:* 429 (March) 1946.
15. Gross, R. E., and Chisholm, T. C.: Annular pancreas producing duodenal obstruction; report of successfully treated case, Ann. Surg. *119:* 759 (May) 1944.
16. Haden, W. D., Jr.: Annular pancreas, Radiology *55:* 859 (Dec.) 1950.
17. Howard, N. J.: Annular pancreas, Surg., Gynec. & Obst. *50:* 533 (March) 1930.
18. Ladd, W. E.: Congenital duodenal obstruction, Surgery *1:* 878 (June) 1937.
19. Lecco, T. M.: Zur Morphologie des Pankreas Annulare, Sitzungsb. d. k. Akad. d. Wissensch. math. naturn Kl. *119:* 391, 1910.

20. Lehman, E. P.: Annular pancreas as clinical problem, Ann. Surg. *115:* 574 (April) 1942.
21. Lerat, P.: Contribution Chirurgicale a l'Etude de pancreas annulaire, Bull Acad. roy. de med. de Belgique, 4th Ser. *24:* 290, 1910.
22. McNaught, J. B.: Annular pancreas; complication of 40 cases with report of new case, Am. J. M. Sc. *185:* 249 (Feb.) 1933.
23. McNaught, J. B., and Cox, A. J., Jr.: Annular pancreas; report of case, with simple method for visualizing duct system, Am. J. Path. *11:* 179 (Jan.) 1935.
24. Nedelec, Stenose duodenale par malformation pancreatique congenitale; sur D2; intervention, Mem. Acad. de chir. *72:* 315 (June 5–12) 1946.
25. Olmacher, A. P., and Marshall, E. A.: Annular pancreas, Am. J. Surg. *79:* 473 (March) 1950.
26. Payne, R. L., Jr.: Annular pancreas, Ann. Surg., *133:* 754 (June) 1951.
27. Ravitch, M. M., and Woods, A. C.: Annular pancreas, Ann. Surg. *132:* 1116 (Dec.) 1950.
28. Silvis, R. S.: Annular pancreas, Ann. Surg. *135:* #2, 278 (Feb.) 1952.
29. Smetana, H.: Ein Beitrag zur Kenntnis der Missbildungen des Pankreas, Beitr. z. path. Anat. u. z. allg. Path. *80:* 239 (Aug.) 1928.
30. Stofer, B. E.: Annular pancreas; tabulation of recent literature and report of case, Am. J. M. Sc. *207:* 430 (April) 1944.
31. Truelsen, F.: Annular pancreas, Nord. Med. (Hospitalstid) *8:* 2226 (Nov. 30) 1940.
32. Vidal, E.: Quelques Case de Chirurgie Pancreatique, Process Verbal Assoc. franc. de chirurgie *18:* 739, 1905.
33. Warren, K. W.: Surgical treatment of uncommon lesions of duodenum, S. Clin. North America *32:* #3 877 (June) 1952.
34. Weissberg, H.: Ein Pancreas annulare bei einem menschlichen Embryo von 16 mm. Lange, Anat. Anz. *79:* 296 (Jan.) 1935.
35. Zech, R. L.: Anomalous pancreas as cause of chronic duodenal obstruction. Report of a case of annular pancreas, West. J. Surg. *39:* 917 (Dec.) 1931.

PARTIAL GASTRECTOMY FOR PEPTIC ULCER

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Of the various operations which have been employed in the treatment of peptic ulcer, only partial gastrectomy has stood the test of extensive clinical trial.[6, 11, 13, 17, 27, 34, 36] Gastrojejunostomy, once the mainstay of American surgeons, has become practically obsolete. The results obtained with vagotomy have been, in general, disappointing.[11, 13, 17] The operation of segmental resection as modified by Wangensteen[34] and which he refers to as "an old operation in a new dress" is based on sound principles. Whether it proves superior to the more conventional types of gastric resection remains to be determined.

Through application of knowledge acquired in the experimental laboratory, the principles of partial gastrectomy have been fairly well defined and the technic of the operation has, accordingly, become somewhat standardized. Nevertheless, a review of the voluminous recent literature on the subject discloses wide variations of opinion regarding some of the indications for partial gastrectomy and certain technical features of the operation.

It is the purpose of this paper to present the policies which I have adopted in regard to partial gastrectomy as illustrated by their application in the management of 109 consecutive cases of peptic ulcer.

PRESENTATION OF CLINICAL MATERIAL

During the period from July 1, 1948, to March 1, 1952, 110 patients were subjected to definitive operations for peptic ulcer on my service at University Hospitals. One patient, a white man 85 years of age with pyloric obstruction due to duodenal ulcer, was successfully treated by gastrojejunostomy. The other 109 patients were treated by partial gastrectomy. These 109 cases constitute the clinical material for this study.

General data. The distribution of cases according to diagnosis, race, sex and age is presented in Tables I and II. Duodenal ulcer was encountered more than twice as frequently as gastric ulcer. The fact that peptic ulcer is predominantly a disease of white men is evident. Although the age of the patients varied from 14 to 76 years, 73.4 per cent were between the ages of 40 and 70 years at the time of operation. Patients with gastric ulcer were an average of 6 5 years older than those with duodenal ulcer.

Indications for operation. The indications for operation as determined preoperatively are listed in Table III. The presence of a gastric lesion was considered an absolute indication for operation. The cases in which the lesion proved to be malignant are not included in this report. Although intractability to medical management was the most common indication in the patients with

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duodenal ulcer, it was the sole indication in only 6 patients. Pyloric obstruction of varying degree was the second most common indication. Hemorrhage of clinically significant severity was an indication in 43 patients. Fourteen of these patients had emergency gastrectomies for massive hemorrhage. Previous acute perforation with subsequent recurrence of ulcer symptoms was an indication in 27 patients. Fourteen patients had delayed primary gastrectomy for acute perforation. A preoperative diagnosis of marginal ulcer was made in 5 patients who had had previous operations for duodenal ulcer. Another patient in whom a

TABLE I

Distribution according to diagnosis, race and sex

Diagnosis	White		Negro		Total
	Men	Women	Men	Women	No. of Cases
Duodenal ulcer	58	9	1	0	68
Gastric ulcer	26	3	0	0	29
Gastric and duodenal ulcer	5	1	0	0	6
Marginal ulcer	5	0	1	0	6
Total no. cases	94	13	2	0	109

TABLE II

Age distribution according to diagnosis

Diagnosis	No. Cases	Age Range	Age Average
		years	*years*
Duodenal and marginal ulcer	74	14–76	49.4
Gastric ulcer	29	28–76	55.9
Gastric and duodenal ulcer	6	46–72	54.1
Entire series	109	14–76	51.1

Age by decades—entire series

10–20	20–30	30–40	40–50	50–60	60–70	70–80	40–70
1	5	16	30	29	21	7	80 (73.4%)

preoperative diagnosis of gastrojejunocolic fistula was made proved to have a gastroileostomy with an ileal ulcer. It is significant that multiple indications were present in 66 per cent of the cases.

Previous operations. Twenty-six per cent of the patients had previously had one or more operations for ulcer or complications of ulcer. The operations are listed in Table IV.

Pathology. Multiple ulcers occurred in 5 of the 35 patients with gastric ulcer (Table V*A*). Although most of the ulcers were located in the prepyloric region or on the lesser curvature, the ulcer in 2 cases was situated on the greater cur-

vature, a site once thought to be reserved almost exclusively for carcinoma. Penetration of the ulcer through the gastric wall into adjacent structures was present in 71.4 per cent of the cases.

Nine of the 74 patients with duodenal ulcer had multiple ulcers (Table V*B*). The ulcers were equally distributed between the anterior and posterior aspects of the first portion of the duodenum. Seven patients had *kissing* ulcers. Narrowing

TABLE III

Indications for operation

Indications	No. of Pts.	Indications Per Patient	No. of Patients
Gastric ulcer	28	1	37 (34.0%)*
Intractability	52	2	49 (45.0%)
Pyloric obstruction	44	3	18 (16.5%)
Previous hemorrhage	29	4	3 (2.8%)
Massive hemorrhage	14	5	2 (1.8%)
Previous perforation	27		
Acute perforation	14		
Marginal ulcer	5		
Gastrocolic fistula	1		

* Distributed as follows: gastric ulcer—21 pts.; intractability—6 pts.; other indications—10 pts.

TABLE IV

Previous operations for ulcer

Operation	No. of Operations	No. of Previous Operations Per Patient	No. of Patents
Closure of perforation	22	None	80
Gastrojejunostomy	6		
Gastrojejunostomy and vagotomy	2	One	22
Vagotomy	3	Two	5
Dismantling of gastrojejunostomy	1	Three	2
Pylorectomy and gastroileostomy	1		
Drainage of subphrenic abscess	3		
Total	38		

29 patients (26.7%) had 38 operations.

of the lumen by cicatrix was present to some degree in over one-half of the cases. In 73 per cent of the cases, the ulcer had penetrated into contiguous structures.

Pathologic features of the 6 cases of marginal ulcer are given in Table V*C*. Penetration of the ulcer through the wall of the intestine was observed in 3 of the cases.

Operations. The operations employed (Table VI) uniformly consisted of resection of 75 to 80 per cent of the stomach, including removal of practically all of the lesser curvature and the proximal portion of the duodenum. The duodenum was transected distal to the ulcer in all except 4 cases. The resections

TABLE V

A. Pathology: gastric ulcer

Total No. Cases	Ulcer, Single	Ulcers, Mult.	Site					Extent of Penetration	
			Lesser Curve	Greater Curve	Pars Media		Pre-pyloric	Limited Stomach	Other Involvement*
					Ant.	Post.			
35	30	5	10	2	2	6	15	10	25 (71.4%)

B. Pathology: duodenal ulcer

Total No. Cases	Ulcer, Single	Ulcers, Mult.	Site			Organic Obstruction		Extent of Penetration	
			Ant.	Post.	Ant. & Post.	Present	Absent	Limited Stomach	Other Involvement†
74	65	9	34	33	7	41	33	20	54 (73%)

C. Pathology: marginal ulcer

Case No.	Previous Operation	Site and Extent of Perforation
1	Gastrojejunostomy	Efferent side of stoma. No perforation
2	Gastrojejunostomy	Efferent side of stoma. Acute perforation
3	Gastrojejunostomy	Stoma. Perforation into mesocolon
4	Gastrojejunostomy and vagotomy	Efferent loop. No perforation
5	Gastrojejunostomy and vagotomy	Efferent loop. Perforation into mesocolon
6	Pylorectomy and gastroileostomy	Ileum. No perforation

* Gastrohepatic omentum (10 cases) Pancreas (8 cases) Hepatoduodenal ligament (4 cases) Gastrocolic omentum (2 cases) Transverse mesocolon, liver and abdominal wall (1 each).

† Pancreas (28 cases) Hepatoduodenal ligament (26 cases) Greater omentum (10 cases) Liver (7 cases) Gallbladder (2 cases).

TABLE VI

Operations

Type	No. of Cases	Ulcer Removed	Ulcer Not Removed	Other Procedures				
				Vagotomy	Repair Incisional Hernia	Cholecystectomy	Resect. Transv. Colon	Appendectomy
Polya.....	51	48	3	0	2	1	0	1
Hoffmeister.....	45*	42*	1	1*	2	2	1	6
Billroth I.....	14	14	0	0	0	1	0	9
Total.....	110	104	4	1	4	4	1	16

* One patient developed marginal ulcer and was treated by re-resection and vagotomy.

for gastric ulcer included removal of the greater omentum, gastrocolic and gastrohepatic omenta and the regional lymph nodes. The spleen and transverse colon were removed also in 1 case with a greater curvature ulcer which was thought grossly to be a carcinoma.

In 96 of the operations, gastrointestinal continuity was reestablishd by retrocolic "no-loop" terminolateral gastrojejunostomy, utilizing the Polya technic in 51 and the modification of Hoffmeister in 45 cases. Gastroduodenostomy by the Schoemaker-Billroth I method was employed in 14 cases.

Auxiliary procedures as listed were performed in 26 cases. One patient who was subjected to a second resection, complemented by vagotomy, will be discussed later.

Postoperative complications and mortality. Ten patients (9.1 per cent) had serious complications (Table VII). The patient, previously mentioned, who had resection of the transverse colon and primary anastomosis of the colon developed a pelvic abscess, probably as a result of inadequate preoperative preparation of the colon. Another patient had a subhepatic abscess due to ischemic necrosis of a portion of the gastrocolic omentum. Pneumonitis was a clinically significant complication in 2 patients, both of who had severe emphysema and pulmonary fibrosis. Two patients, one of whom had had extensive dissection of the abdominal

TABLE VII

Postoperative complications and mortality

Complications	No. Cases	Deaths
Pelvic abscess	1	0
Subhepatic abscess	1	0
Pneumonitis	2	0
Wound infection	2	0
Wound disruption	2	1*
Bleeding from stoma	2	0
Total	10 (9.1%)	1 (0.9%)

* W. M., 71, emergency gastrectomy for massive bleeding from duodenal ulcer. Disruption on 8th day. Sudden death 10th day. Autopsy not permitted.

wall for repair of a large incisional hernia, had wound infections. Dehiscence of the wound occurred in 2 cases. Bleeding from the anterior portion of the gastroenteric stoma, occurred in 2 patients due to improper placement of the Connell suture. In both instances, excessive blood loss was prevented by prompt recognition of the condition and immediate operation.

There was one death in the series. The patient, a white man 71 years of age, died 10 days after emergency gastrectomy for a bleeding duodenal ulcer and two days following resuture of a disrupted wound. Pulmonary embolism was thought to be the cause of death.

Results. As shown in Table VIII, 107 patients have been followed for periods varying from three months to four years. The follow-up has been conducted by personal interview and physical examination at regular intervals and by annual questionnaires. Although all patients have had one determination of gastric acidity and one roentgenographic examination, additional studies have been limited to the patients who have or have had symptoms.

All of the patients state that they are satisfied with the operation. Eighty-eight (82.2 per cent) are entirely free of symptoms and dietary restrictions and are considered as having excellent results (Table IX). Seventeen patients (15.9 per cent) are well but have minor annoying symptoms as listed in Table X. Ten of these patients have *dumping syndrome* which is minimized or prevented entirely by avoiding concentrated carbohydrates and/or large volumes of liquid in the diet. Four patients have occasional vomiting of bile and 3 others complain of not being able to eat a full sized meal because of a sensation of excessive fullness. Some of the patients included in the excellent group had similar symptoms in the early postgastrectomy period which subsequently disappeared.

Unsatisfactory results were obtained in 3 patients. Two of these are listed as having fair results. Both have severe symptoms (Table X), yet they insist that they are satisfied with the operation. The patient whose chief complaints are nausea and anorexia has complete antacidity and is believed to have atrophic

TABLE VIII

Duration of follow-up

No. in Series	Died	Follow-up		Duration of Follow-up			
		Lost	Available	Under 1 yr.	1–2 yrs.	2–3 yrs	Over 3 yrs.
109	1	1	107	29	40	26	12

TABLE IX

Results

Result	No. Cases	Result	No. Cases
Excellent	88 (82.2%)	Fair	2 (1.8%)
Good	17 (15.9%)	Poor	1 (0.9%)*
Total satisfactory	105 (98.1%)	Total unsatisfactory (corrected)	2 (1.8%)

* Patient who developed marginal ulcer converted to excellent result after re-resection and vagotomy.

gastritis. The symptoms of the other patient are difficult to evaluate because of his psychotic traits.

One patient in the excellent group was initially classified as having a poor result. This patient, a white woman 42 years of age, was operated upon because of recurring episodes of hemorrhage and increasingly severe obstruction due to a large duodenal ulcer. Hyperacidity persisted after resection. Three months later, she reentered the hospital because of recurrent pain and moderately severe bleeding. At operation a penetrating marginal ulcer was found. Re-resection and subdiaphragmatic vagotomy were performed. She has remained well during the ensuing nine months.

ACUTE PERFORATION AND MASSIVE HEMORRHAGE

The methods employed in the management of patients with acute perforation and massive hemorrhage warrant special comment. The types of treatment for

acute perforation which are currently in vogue include surgical closure of the perforation,[32, 33] nonoperative treatment,[4] and primary gastrectomy.[3, 33] The criteria used for the application of these methods vary considerably. Realizing the significance of perforation as an indication of virulence, many surgeons now advocate definitive surgery for all patients who have had acute perforations.[33] In order to expedite the attainment of that goal, I have, since February 1949, routinely performed partial gastrectomy in the early postperforation period. This plan of delayed primary gastrectomy[3] has been followed in the treatment of

TABLE X

Postgastrectomy symptoms

	No. of Patients
A. Patients classified as having good results:	
Mild dumping, controlled by diet	10
Occasional vomiting (1 to 3 times a month)	4
Inadequate capacity (4 to 6 meals a day)	3
B. Patients classified as having fair result:	
Nausea and anorexia. Many unrelated symptoms. Unable to work. Draws old-age pension	1
Occasional vomiting. Weakness. Unrelated complaints. Alcoholism and severe psychoneurosis requiring psychiatric care before and since operation	1
C. Patient classified initially as having poor result:	
Recurrent pain and bleeding 3 months after operation. Well 9 months after re-resection and vagotomy	1

TABLE XI

Delayed primary gastrectomy for acute perforation

Number of cases (duodenal 11; gastric 2; marginal 1)	14
Average delay, perforation to gastrectomy (range, 4–26 days)	13.3 days
Surgical closure as part of initial treatment	2 cases
Massive hemorrhage after perforation	2 cases
Complication (wound disruption)	1 case
Mortality rate	0

14 patients with acute perforation (Table XI). The method has proved safe, sound in principle and applicable to all cases.

The treatment of massive hemorrhage is certainly not standardized. Some surgeons advocate operation in all cases,[31] others only in selected cases or under certain circumstances[1, 2, 12, 15, 29] and still others, in no cases.[7] There are even differences of opinion regarding the definition of massive hemorrhage. Despite conclusive clinical and experimental evidence of the desirability of rapid restoration and maintenance of an adequate blood volume[20, 31] there are those who still attempt to treat hemorrhage by maintaining shock.

Fourteen patients in this series had massive hemorrhage (Table XII). Attempts to exclude other causes of bleeding by barium studies were necessary in

6 cases. The method of management was patterned after that of Stewart, et al.[31] Treatment consisted of restoration of blood volume by rapid transfusion of whole blood and operation as soon as there was clinical evidence of response to shock therapy. Operation under such circumstances should be considered as an integral part of the resuscitation since its primary purpose is to arrest hemorrhage. Definitive treatment of ulcer by partial gastrectomy is essential for preventing recurrence of bleeding, but it can be postponed for several days if necessary. Gastric resection was accomplished at the initial operation in all of the cases in this group. The operation was performed on 12 patients within 12 hours after their admission. Temporary cessation of bleeding led to delays of 36 and 72 hours, respectively, in 2 cases.

Data pertaining to the one death which occurred in this group of cases are summarized in Table VII. The relatively low mortality rate of 7.1 per cent demonstrates the value of aggressive treatment of massive hemorrhage. The experimental observations of Le Veen, et al.[20] supports the validity of this method.

TABLE XII

Emergency gastrectomy for massive hemorrhage

Number of cases (duodenal 10, gastric 3, marginal 1)	14
Average duration on admission (range, 4 hours to 14 days)	4.7 days
Average delay, admission to operation (range, 1 to 72 hours)	13.4 hours
Average hemoglobin on admission (range, 3 to 9 grams)	6.5 grams
Complications; subphrenic abscess 1: wound disruption 1 (fatal)	2 cases
Mortality rate	7.1 per cent

DISCUSSION

Because of the high incidence of carcinoma in gastric lesions which are thought clinically to be benign,[18, 30] gastric ulcer should be considered a surgical disease. Fortunately, the results of resection for gastric ulcer are almost invariably satisfactory. Duodenal ulcer, however, is a different disease and the results of surgical treatment are not always acceptable to the patient, particularly, if operation is undertaken in the absence of definite indications for surgical intervention. If any single factor is primarily responsible for the high incidence (98.1 per cent) of satisfactory results in this series of cases, it is the rigidity with which adherence to recognized indications for operation was maintained. Most of the patients had multiple indications for operation and exhibited advanced pathologic changes including penetration of the ulcer into adjacent structures in over 70 per cent of the cases. It should be emphasized that *intractability* which can not be explained by the severity or extent of the pathologic process is a poor indication for operation.

The technical aspects of partial gastrectomy for duodenal ulcer which are the principal subjects of controversy at present are those pertaining to the management of the duodenum and to the method of restoration of gastrointestinal continuity. Colp[6] states that while excision of the ulcer is highly desirable, it is not essential as long as the pylorus is completely removed. This opinion is

shared by Eastman and Cole,[9] Glenn and Harrison,[14] and Miller and Ripstein.[22] Although the shortcomings of the Finsterer operation for antral exclusion are generally appreciated, the procedure is occasionally employed in selected cases by some surgeons.[11, 23] Ross and Warren[26] and Glenn and Harrison[14] agree that when such a compromise procedure is necessary, it should be followed by removal of the ulcer at a later time. Lewisohn[21] contends that a modified Finsterer operation is frequently done inadvertently by surgeons who leave the ulcer in situ and, accordingly, if the ulcer is not excised, the operation should be labelled a "palliative" resection.

In all except 4 of the cases in this series, the ulcer was excised. The dangers of resecting an ulcer of the duodenum in the presence of acute inflammation have been emphasized by others.[2, 14] Nevertheless, the experience with delayed primary gastrectomy for perforated ulcer has demonstrated that the edema actually facilitates dissection of the duodenum and that removal of the ulcer in such cases is generally less hazardous than it is in the presence of extensive chronic inflammation. In cases of the latter type, in which penetration of the duodenal wall has produced marked periduodenal fibrosis, damage to the pancreas or common duct can be avoided by opening the duodenum at the upper border of the ulcer and freeing the duodenum around the crater by meticulous dissection under direct vision. The portion of the ulcer crater which is extrinsic to the duodenum need not be disturbed.

Insertion of a drain down to the duodenal stump has been advocated in cases where there is doubt as to the security of the duodenal closure.[6, 37] Drains were not used in any of the cases in this series, reliance being placed on careful technic to prevent leakage and close observation to detect it. No instances of leakage were encountered. One patient who exhibited the classical syndrome[19] on the third postoperative day was found, on exploration, to have necrosis of a portion of the gastrocolic omentum.

The most popular type of reconstruction after gastric resection is the retrocolic *no-loop* gastrojejunostomy, which is generally considered to be more physiologic, less conducive to functional disorders and less inclined to marginal ulceration than antecolic anastomosis.[11, 14, 22, 26, 34, 37] Others,[6, 23] however, claim equally good results with antecolic anastomosis and prefer that type because of its simplicity. In this series, all of the operations in which continuity was established by gastrojejunostomy were of the retrocolic type, the Polya and Hoffmeister technics being employed with about equal frequency and with equally good results. The Schoemaker-Billroth I operation,[5, 16, 28] which was used in 14 cases, is simpler, safer, more physiologic and can be accomplished more rapidly than the Polya or Hoffmeister operation. Unfortunately, however, it is not anatomically feasible in all cases. Perhaps it can be applied in a greater proportion of cases by use of the Finney modification as currently employed by Fallis.[10]

Numerous variations of the technic of partial gastrectomy have been advocated as ways to prevent or to decrease the incidence of the *dumping syndrome*. Among these are the production of a small stoma,[25] the fashioning of a valve at the afferent side of the stoma,[22] antecolic anastomosis with the afferent loop on

the greater curvature,[24] and the provision of additional peritoneal support of the gastric remnant.[35] Schoemaker[28] stated that *dumping* did not occur in the cases in which he had employed the Schoemaker-Billroth I operation, yet several of his patients had symptoms that are usually considered a manifestation of *dumping*. One of the 14 patients in this series who had that type of operation has the syndrome.

Until the mechanism of *dumping* is more definitely established it is not likely that the syndrome will be completely eliminated. Fortunately, when it does occur, it is often transient. If not, it usually persists in a mild form. As a rule, the patient so afflicted readily accepts the disturbance as a small price to pay for restoration of his health and economic independence.

SUMMARY

An analysis of 109 patients with peptic ulcer who were treated by partial gastrectomy is presented. The incidence of serious complications following operation was 9.1 per cent. The mortality rate was 0.9 per cent, the single fatality occurring in the case of an elderly man subjected to emergency gastrectomy for massively bleeding duodenal ulcer.

One patient developed marginal ulcer after gastric resection. She is well nine months after re-resection and vagotomy. The results in 2 patients who have severe postgastrectomy symptoms is considered unsatisfactory. Seventeen patients (15.9 per cent) are well but have mild functional disturbances. Eighty-eight patients (82.2 per cent) are entireyl free of symptoms and dietary restrictions. Satisfactory results were obtained in 98.1 per cent of the cases.

The currently popular methods of management of acute perforation and massive hemorrhage are summarized. The methods employed in the treatment of 14 patients with acute perforation and 14 patients with massive hemorrhage are presented.

The indications for surgical treatment of peptic ulcer and certain controversial matters pertaining to the technic of partial gastrectomy are discussed.

CONCLUSIONS

Partial gastrectomy for peptic ulcer can be accomplished with a low mortality rate and a relatively low morbidity rate.

The results of partial gastrectomy for gastric ulcer are almost invariably satisfactory. Equally good results can be obtained when the operation is done for duodenal ulcer if proper care is exercised in the selection of candidates for surgical treatment.

Delayed primary gastrectomy is a safe, sound and widely applicable method for the treatment of peptic ulcer with acute perforation.

In the management of massive hemorrhage due to peptic ulcer, rapid restoration of blood volume by whole blood transfusions is essential. Emergency operation to control hemorrhage and to prevent recurrence by providing definitive treatment for the ulcer should be considered an integral part of the resuscitation.

As long as the basic requirements of partial gastrectomy for peptic ulcer are satisfied, the manner in which gastrointestinal continuity is restored is relatively unimportant.

REFERENCES

1. Amendola, F. H.: The management of massive gastroduodenal hemorrhage, Ann. Surg. *129:* 47 (Jan.) 1949.
2. Amendola, F. H.: Surgical intervention in massive gastroduodenal bleeding, Surgery *31:* 340 (Mar.) 1952.
3. Auchincloss, H., Jr.: Immediate sub-total gastrectomy for acute perforated peptic ulcer, Ann. Surg. *135:* 134 (Jan.) 1952.
4. Bedford, T. E. W.: Conservative treatment of perforated duodenal ulcer, Brit. M. J. *1:* 457 (Mar. 31) 1945.
5. Castleton, K. B.: The Billroth I type of gastric resection, American Surgeon *18:* 546 (June) 1952.
6. Colp, R.: Surgical management of duodenal ulcer, Surg., Gynec. & Obst. *91:* 306 (Sept.) 1950.
7. Costello, C.: Massive hematemesis, Ann. Surg. *129:* 289 (Mar.) 1949.
8. Dodson, H. C., Jr.: Delayed primary gastrectomy for perforated peptic ulcer, Ann. Surg. *135:* 139 (Jan.) 1952.
9. Eastman, W. H. and Cole, W. H.: Precautions and results in gastrectomy, Arch. Surg. *59:* 768 (Sept.) 1949.
10. Fallis, L. S.: Personal communication.
11. Ferguson, L. K., Sala, L. F., and Shaver, W. A.: Gastrectomy for peptic ulcer, S. Clin. North America *31:* 1693 (Dec.) 1951.
12. Frazer, R. W. and West, J. P.: The management of bleeding duodenal ulcers, Ann. Surg. *129:* 299 (Mar.) 1949.
13. Glenn, F.: Present status of the surgical treatment of peptic ulcer, J.A.M.A. *145:* 790 (Mar. 17) 1951.
14. Glenn, F. and Harrison, C. S.: Gastric resection in the treatment of peptic ulcer, Arch. Surg. *62:* 151 (Feb.) 1951.
15. Glenn, F. and Harrison, C. S.: Surgical management of massive hemorrhage from peptic ulcer, Arch. Surg. *63:* 766 (Dec.) 1951.
16. Higginson, J. F. and Clagett, O. T.: Gastric resection; Schoemaker-Billroth I operation, Surgery *24:* 613 (Oct.) 1948.
17. Jordon, S. M.: Report of the chairman of the subcommittee on surgical procedures in peptic ulcer of the American gastroenterological association, Gastroenterology *19:* 599 (Dec.) 1951.
18. Lampert, E. G., Waugh, J. M., and Dockerty, M. B.: The incidence of malignancy in gastric ulcers believed preoperatively to be benign, Surg., Gynec. & Obst. *91:* 673 (Dec.) 1950.
19. Larsen, B. B. and Foreman, R. C.: Syndrome of the leaking duodenal stump, Arch. Surg. *63:* 480 (Oct.) 1951.
20. LeVeen, H. H., Mulder, A. G., and Prokop, F.: The physiological mechanism for death in massively bleeding peptic ulcer, Surg., Gynec. & Obst. *94:* 433 (Apr.) 1952.
21. Lewisohn, R.: Basic principles in the surgical treatment of duodenal ulcers, J.A.M.A. *149:* 423 (May 31) 1952.
22. Miller, G. G. and Ripstein, C. B.: Technique of gastric resection for peptic ulcer, Surg., Gynec. & Obst. *89:* 464 (Oct.) 1949.
23. Nicolaysen, K. and Fretheim, B.: Partial gastrectomy for ulcer and postoperative complications, Surgery *30:* 597 (Oct.) 1951.
24. Parker, J. M.: Personal communication.
25. Porter, H. W. and Claman, Z. B.: A preliminary report on the advantages of a small stoma in partial gastrectomy for ulcer, Ann. Surg. *129:* 417 (Apr.) 1949.
26. Ross, F. P. and Warren, R.: Safeguards in gastric resection for duodenal ulcer, New England J. Med. *245:* 475 (Sept. 27) 1951.
27. St. John, F. B., et al.: Results following subtotal gastrectomy for duodenal and gastric ulcer, Ann. Surg. *128:* 3 (July) 1948.
28. Schoemaker, C. A.: Partial gastrectomy in the war years, Surg., Gynec. & Obst. *88:* 447 (Apr.) 1949.
29. Shapiro, N. and Schiff, L.: Ten years' experience with bleeding peptic ulcer with emphasis on forty-five fatal cases, Surgery *31:* 327 (Mar.) 1952.
30. Starry, L. J. and Dodson, H. C., Jr.: Carcinoma of the stomach, Jour. Internat. Coll. Surgeons *17:* 417 (Apr.) 1952.

31. Stewart, J. D., et al.: Management of massively bleeding peptic ulcer, Ann. Surg. *128:* 791 (Oct.) 1948.
32. Taylor, F. W. and Egbert, H. L.: Nonoperative treatment of perforated peptic ulcer, Surg., Gynec. & Obst. *94:* 464 (Apr.) 1952.
33. Turner, F. P.: Acute perforations of stomach, duodenum and jejunum, Surg., Gynec. & Obst. *92:* 281 (Mar.) 1951.
34. Wangensteen, O. H.: Segmental gastric resection for peptic ulcer, J.A.M.A. *149:* 18 (May 3) 1952.
35. Welbourn, R. B., Butler, T. J., and Capper, W. M.: Discussion on postgastrectomy syndromes, Proc. Roy. Soc. Med., London *44:* 773 (Mar. 27) 1951.
36. Wellington, J. S., Brizzolara, L. G., and Silvani, H. L.: Clinical experience with gastric resection in the treatment of peptic ulcer, West. J. Surg. *59:* 648 (Dec.) 1951.
37. Woodruff, R.: Standardization of gastric resection in the treatment of peptic ulcer, American Surgeon *18:* 586 (June) 1952.

DIVERTICULITIS OR CARCINOMA OF THE COLON?

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The difficulty of differentiating carcinoma from diverticulitis has been appreciated for many years. In 1898, Graser[3] pointed out that obstruction was frequent and that the stenosing obstructing lesion caused by inflammation in diverticula could mimic cancer of the colon.

There are several facets to the problem; *carcinoma or diverticulitis.* Cancer of the colon may simulate diverticulitis, diverticulitis may simulate cancer or the two may coexist with signs and symptoms suggesting the presence of only one entity. It is this latter problem which furnishes the greatest diagnostic and therapeutic difficulty.

The history of the relationship of diverticulitis to carcinoma is interesting. In 1911, Wilson[10] emphasized the relationship of diverticulitis to carcinoma and stated that chronic inflammation predisposed the mucosa to the later development of cancer. This view was held by various authors until 1930 when Rankin and Brown[5] refuted it. Their findings have been supported by much valid statistical data. In 1951, Rowe and Kollmar[7] reported that a review of the literature through May 1950 had revealed only 62 definite cases of coexisting carcinoma and diverticulitis of the large bowel. These authors in their excellent article added 7 additional cases from their own records and the records of local hospitals. They stated that doubtless many cases of coexisting diverticulitis and carcinoma had not been reported.

Stimulated by several colon lesions in which coexisting disease was found and in which differentiation was most difficult, a study had been made of all patients having diverticulitis and carcinoma of the colon admitted to the surgical service of the St. Louis City Hospital over a 12 year period.

Table I shows the results of a survey made covering the 12 year period, 1939 to 1951. Two hundred fifty-nine patients with carcinoma of the large bowel were admitted to the St. Louis City Hospital. During the same 12 year period, 30 patients with diverticulitis were admitted. Twenty-five patients were found to have coexisting carcinoma and diverticula while 6 patients were found to have both carcinoma and diverticulitis, 4 of these in the same segment of bowel (rectosigmoid). It will be seen (Table II) that 25 patients had diverticula and carcinoma in the same segment. In reviewing the hospital charts, it was found, on five occasions, that the original impression of diverticulitis was pathologically not proved and that it was actually neoplasm that was causing the signs and symptoms preoperatively attributed to diverticulitis. Thus the chance of diagnostic error was real but the cases are not included as diverticultis because histologic evidence of inflammation was not present.

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Of the 259 cases of carcinoma of the colon, there were 4 cases of diverticulitis in the same segment or 1.54 per cent. These figures agree with previous reports and emphasize that the relationship between cancer of the colon and diverticulitis is coincidental.

The primary problem is diagnostic. The surgeon seeing the patient with diverticulitis of the colon complicated by abscess, obstruction and/or fistula formation is faced with the question, *is it diverticulitis, or cancer, or both?* That the problem is a real one can be best illustrated by the following case reports on patients seen and operated upon by one of us in the past year.

TABLE I

1939 to 1951

DIAGNOSIS	NO. OF CASES
Carcinoma Colon	259
Diverticulitis Colon	30
Carcinoma and Diverticulosis	25
Carcinoma and Diverticulitis	6

TABLE II

1939 to 1951

DIAGNOSIS	NO. OF CASES
Carcinoma Rectosigmoid	111
Diverticula and Carcinoma Rectosigmoid	25
Diverticulitis and Carcinoma Rectosigmoid	4

Case No. 1. A 67 year old white man entered the third surgical division of the St. Louis City Hospital on August 24, 1952. His complaints were weakness, abdominal pain, weight loss and nocturia three to four times nightly without dysuria. He stated that on several occasions he had passed bright red blood per rectum. Significant findings on examination were anemia, a red blood count of 3,000,000 per cu. mm., hemoglobin 7 Gm. per 100 cc. and the presence of left lower quadrant tenderness on palpation. The patient had a known cardiac condition with previous admissions to the City Hospital because of arteriosclerotic heart disease. Sigmoidoscopy up to 9 cm. revealed visible diverticula, edema of the mucosa and marked tenderness. Because of the edema it was impossible to pass the sigmoidoscope further. No tumor masses were seen. Barium enemas were given on four occasions. The roentgenologic department at each examination (fig. 1) reported numerous diverticula of the sigmoid and distal descending colon with associated tenderness and spasm suggesting inflammatory disease. Cystograms revealed a fistula between the colon and the bladder which could not be demonstrated by cystoscopy since the bladder could not be filled. Two weeks after admission a transverse colostomy was done to divert the fecal stream from the area of diverticulitis and sigmoido-vesicle fistula. Six weeks after the colostomy, and after preparation of the bowel with aureomycin, celiotomy was done. A large, firm fixed mass, involving the midportion of the sigmoid colon was found. This mass was adhered to the bladder and a fistula between the sigmoid colon and urinary bladder was present. Numerous diverticula showing chronic inflammation were present.

Specimens for biopsies were taken from the mass, from the fistula and from within the

lumen of the sigmoid. These were reported to be benign on frozen section. Accordingly, an anterior resection of the colon was done with primary anastomosis. It was impossible to close the large fistulous opening into the bladder, therefore an isolated ileal pouch bladder was constructed and the ureters were transplanted into the ileal segment. The ileal stoma was brought out through a right lower quadrant incision. At the completion of the operation, the pathologist reported that further examination of the material submitted revealed carcinoma. The final pathological report was adenocarcinoma of the sigmoid colon, diverticulitis, adenocarcinoma in the tissue from the bladder fistula. Two weeks later the patient was reoperated upon with the intention of doing a pelvic evisceration. A small nodule on the mesentery of the small bowel was found to be metastatic adenocarcinoma. Accordingly,

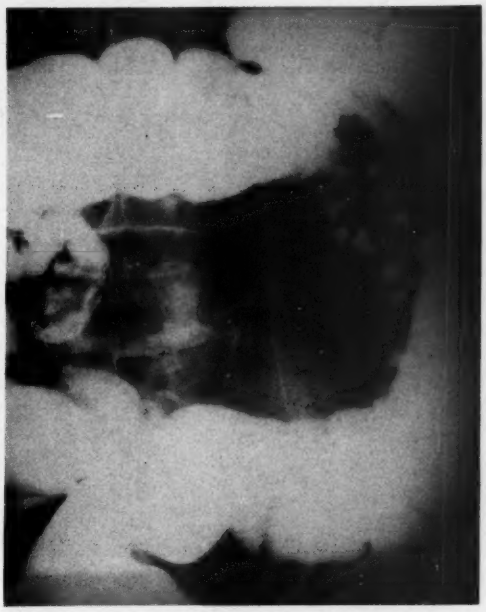

FIG. 1. The roentgenologist reported numerous diverticula of the sigmoid and distal descending colon. Associated tenderness and spasm suggested inflammatory disease.

the patient was considered to be inoperable. The colostomy was closed. This patient died three months later. The cause of death was arteriosclerotic heart disease and recurrent carcinoma.

Comment. This case demonstrates the pitfalls of diagnosis from the standpoint of history, physical findings, sigmoidoscopy, roentgenologic diagnosis and gross findings at operation. While the outcome was fatal, due to his advanced disease, a period of six months to one year's delay after enterostomy would have been most embarrassing in view of the pathological diagnosis.

Case No. 2. A 65 year old white woman entered the St. Louis City Hospital, January 6, 1952. She was acutely ill, complaining of nausea and vomiting and severe generalized abdominal pain. She stated that she had had recent weight loss, constipation and blood in the stools. Examination revealed a markedly distended abdomen showing generalized rigidity and rebound tenderness in all quadrants. Bowel tones were absent. Laboratory examination revealed a red blood count of 3,250,000 per cu. mm. and hemoglobin 12 Gm. per 100 cc.

Roentgenogram on January 6 showed free air under both diaphragms and a small and large bowel pattern compatible with paralytic ileus.

The abdomen was opened on January 7 and found to contain purulent material and feces. A transverse loop colostomy was done. The abdomen was not explored. With careful supportative treatment, the postoperative course was uneventful. On February 1, barium enema studies from below and through the distal colostomy loop were done (fig. 2). The roentgenologists' interpretation was diverticulitis of the sigmoid with perforation and obstruction. Sigmoidoscopy to 18 cm. was negative. The patient was prepared with intestinal

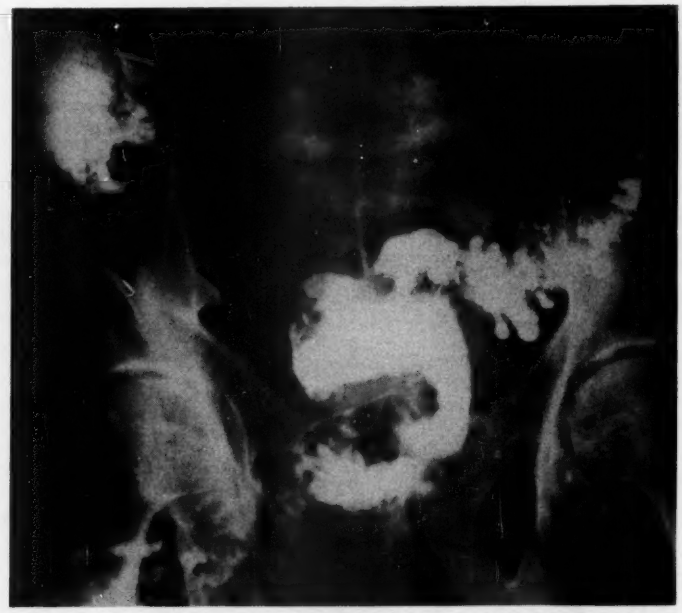

FIG. 2. The roentgenologist diagnosed diverticulitis of the sigmoid with perforation and obstruction.

antibiotics and blood replacement for resection and on February 13, an exploration was done. At operation a metastatic carcinoma was found in the liver and the mesentery of the small bowel. The primary lesion was found in the sigmoid, which was bound down but it was resectable. Because of the liver metastasis and peritoneal implants, and since a decompression colostomy had been done, resection was decided against. The patient is at present time rapidly growing worse, two months after operation.

Comment. This patient presented a rather typical picture of perforation of diverticulitis with resulting peritonitis. A coexisting carcinoma was suspected because of the anemia and weight loss but a definite diagnosis could not be made prior to exploration. This was unfortunately an inoperable lesion.

DISCUSSION

The symptom of carcinoma and diverticulitis both have been well defined and little of a positive character by perusal of individual case records can be expected to be added. The value of study of records is to emphasize that no re-

liance can be placed on symptomatology or physical signs in an individual case, although symptoms such as bleeding and cachexia are most suspicious of carcinoma. This applies also to sigmoidoscopic examination and roentgenologic findings. These adjuncts are valuable but occasionally the diagnosis can be made only in the pathological laboratory by examination of the resected specimen. Since diverticulitis usually becomes a surgical problem in the age group in which carcinoma is most frequent, and since both lesions may produce obstruction, mass and bleeding with a high degree of regularity in the same segment of large intestine, it is understandable that differentiation is difficult. An analysis of all records shows that bleeding of some degree occurred in 62 per cent of the carcinomas and in 4 of 30 cases of diverticulitis or 18.8 per cent. In no case of diverticulitis was the bleeding massive. While bleeding can and does occur with diverticulitis, as reported by Rosser[6] and Fansler[2], the presence of blood places an additional burden of proof of the surgeon to rule out the presence of cancer. Massive hemorrhage from diverticulitis has been reported by Turnbull[9] and is explained by Lahey[4] on the basis of rupture of a nutrient vessel of an epiploic appendix adjacent to the sac of an eroded diverticulum.

Change in bowel habits occurred in both diseases with regularity. Pain and tenderness occurred in 58 per cent of the patients with diverticulitis, in 16 per cent of those patients with carcinoma of the rectosigmoid and 12 per cent of all carcinomas of the colon. However, in patients with coexisting diseases, pain and tenderness were present in all cases, making this of no value in differentiation.

Urinary symptoms occur in those cases in which the colon perforates adjacent to or into the bladder. Six patients of our series had fistulas between the colon and the urinary bladder. Two of these patients had cancer alone, 2 had diverticulitis alone and 2 had coexisting disease. It thus becomes apparent that while generalizations can be made, signs and symptoms furnish only presumptive evividence in an attempt to differentiate the pathological processes.

Sigmoidoscopy is, of course, of great value when the lesions can be seen and a specimen for biopsy can be taken. If, however, the lesion is out of reach of the sigmoidoscope, accurate differentation cannot be made. Rowe and Kollmar cite the work of Wisseman, Lemon and Lawrence[11] in applying the Papanicolaou technic to the diagnosis of cancer of the colon and refer to a case of their own in which rectal smears containing atypical cells were obtained. This may be an aid in differentiation and continued trial with this method is justified. We have had no experience with it.

The value of the roentgenogram in diagnosis has long been known and is the most important aid we have for differentiation with the exception of a biopsy. Schatzki[8] points out that differentiation is possible in a fair precentage of cases. He emphasizes the importance of preservation of the mucosal folds and stresses that the area of colon above and below malignant growths appears normal. While the roentgenogram admittedly is of value, we present here a rather typical roentgenogram of 1 of our patients who had coexisting disease. This was used preoperatively on a university hospital service as a teaching roentgenogram to illustrate many of the points typical of diverticulitis (fig. 3).

It is obvious that some confusion will persist in diagnosis despite close attention to all differential diagnostic possibilities. How then can we give rational and safe treatment in view of uncertain diagnosis? We must assume that any case of complicated diverticulitis harbors the possibility of associated cancer. The literature is replete with the difficulty of separating benign from malignant lesions, yet attention often is directed to the danger of resecting diverticulitis under the mistaken impression that one is dealing with cancer. This idea originated at a time when colon surgery was fraught with high morbidity and mortality.

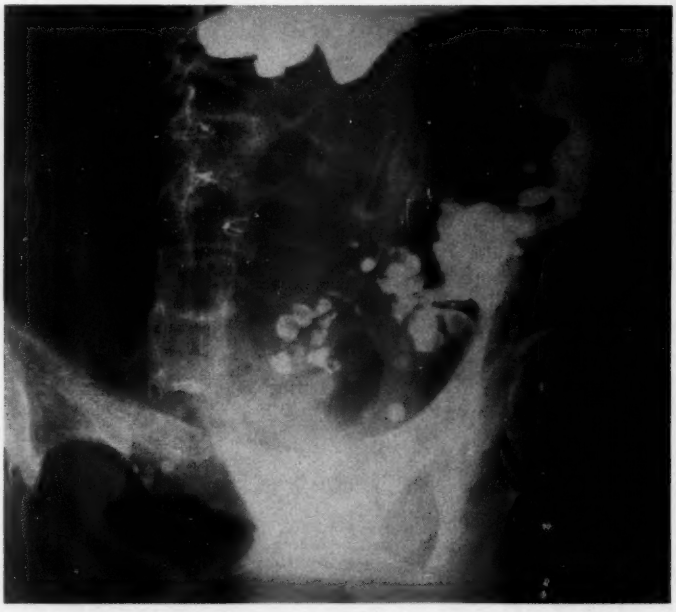

FIG. 3. This roentgenogram was used in teaching medical students to show them the typical appearance of diverticulitis. Carcinoma was present but was not diagnosed.

Since the advent of more perfect anesthesia, adequate blood replacement, efficient intestinal antibiotics and gastrointestinal tract decompression by means of the indwelling tube, the picture of colon surgrey has changed. The wisdom of treating the surgical complications of diverticulitis by colostomy followed in six months to one year by a resection must, in the light of present knowledge, be questioned. By such therapy a burdensome, unnecessarily protracted, unpleasant, expensive procedure is carried out.

We make no claim to originality in recommending early resection. This view has been stressed in the past by Rosser[5] and Cattell[1] and especially by Rowe and Kollmar. We believe, however, that the consensus of surgical thought is still that of long waiting pericds after colostomy before resection. We believe that necessary decompression measures should be carried out and that they are appli-

cable to either carcinoma or diverticulitis. The most important fact is that plans for definitive treatment should be accelerated.

Thus the modern therapy of complicated diverticulitis should proceed promptly to definitive treatment. This treatment being early resection, which is good therapy for diverticulitis, has the additional advantage of avoiding the pitfall of temporizing with coexisting carcinoma. This is another example how modern technical advances have required us to discard what formerly were regarded as principles. With the minimizing of complications by appreciation of the factors outlined previously, we can approach treatment more logically.

In the last 8 patients with diverticulitis of the sigmoid colon seen on the surgical service of the St. Louis City Hospital, resection was done early in all cases without mortality. Six of these patients had perforated or acutely obstructed colons and proximal colostomy was done followed by anterior resection in periods of from 3 to 12 weeks. The remaining 2 patients had intractable, partially obstructing diverticulitis. These patients, after adequate bowel preparation, had colon resection in one stage without complementary colostomy.

Our specific method of management of diverticulitis is as follows: In those patients with acute conditions, with abscess or perforation, loop colostomy, preferably in the right transverse colon, is done. We stress the importance of right colostomy as we believe it sets the stage for more complete and adequate left colectomy should a neoplasm be discovered. We have long been advocates of complete left hemicolectomy for carcinoma of the left colon with anastomosis of the midtransverse colon to the distal rectal stump and sacrifice of the inferior mesenteric artery at its point of origin from the aorta. It is for this reason that we often prefer cecostomy, if feasible, as a decompresisve measure. Following proximal colostomy, resection is advocated within a period of two to six weeks. In the absence of abscess, perforation or obstruction, anterior resection is recommended after adequate chemotherapeutic preparation of the large bowel. We have recently been pleased with neomycin as an agent for bowel sterilization. Continuous spinal anesthesia is preferred. Steel wire abdominal closure has been most valuable and had reduced the possibility of dehiscence and evisceration in those patients who are malnourished and potentially infected.

A more aggressive approach to the surgical complications of diverticulitis seems warranted. This is made practical by the tremendous strides which have been made in the development of colon surgery. While diverticulitis coexisting with carcinoma is a coincidence, the possibility is ever present and differentiation is difficult or may be impossible. The imposition of long waiting periods after proximal colostomy in patients with diverticulitis is incapacitating, expensive, uncomfortable, and may be dangerous.

SUMMARY

Diverticulitis of the colon is difficult to differentiate from cancer on the basis of symptoms, signs and operative findings. This diagnostic confusion has in the past led to unfortunate delay in definitive treatment. This delay has been dictated by fear of surgical complications.

Our cases have been reviewed and 6 additional cases of coexisting carcinoma and diverticulitis of the colon have been added to the cases recorded in the literature.

We recommend a more aggressive attitude in the surgical treatment of complicated diverticulitis of the colon, especially when coexistent carcinoma is a possibility.

REFERENCES

1. Cattell, R. B.: Inflammatory stenosing disease of the bowel. Presented before the Dallas So. Clinical Soc., March 1949.
2. Fansler, W. A.: Diverticulitis and diverticulosis; with particular reference to large bowel, Tr. Am. Proct. Soc. *41:* 231, 1940.
3. Graser, E.: Entzundliche Stenose des Dickdarmes bedingt durch Perforation Multipler falscher Divertikel, Centralbl. f. Chir. *26:* 140, 1898.
4. Lahey, F.: Advantages of an hepatic flexure mikulicz type of defunctionalizing colostomy in surgical treatment of diverticulitis, Lahey Clin. Bull. *6:* 34 (Oct.) 1948.
5. Rankin, F. W., and Brown P. W.: Diverticulitis of colon, Surg., Gynec. & Obst. *50:* 836 (May) 1930.
6. Rosser, C.: Surgical management of diverticulitis, South M. J. *32:* 1203 (Dec.) 1939.
7. Rowe, R. J., and Kollmar, G. H.: Diverticulitis of the colon complicated by carcinoma, Internat. Abst. Surg. *94:* 1 (Jan.) 1952.
8. Schatzki, R.: Roentgenologic differential diagnosis between cancer and diverticulitis of colon, Radiology *34:* 651 (June) 1940.
9. Turnbull, G. C.: Massive hemorrhage from diverticula of the colon; report of 12 patients, Quart. Bull. Northwestern Univ. M. School *22:* 292, 1948.
10. Wilson, L. B.: Diverticula of the lower bowel; their development and relationship to carcinoma, Ann. Surg. *53:* 223 (Feb.) 1911.
11. Wisseman, C. L., Jr., Lemon, H. M., and Lawrence, K. B.: Cytologic diagnosis of cancer of descending colon and rectum, Surg., Gynec. & Obst. *89:* 25 (July) 1949.

THE CLINICAL SIGNIFICANCE OF PROLAPSED GASTRIC MUCOSA

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The significance which should be attributed to a given condition must be determined by the accumulated clinical experience of competent observers over an adequate period of time. While criteria for the recognition of the entity must be established, and attention devoted to the development and selection of appropriate methods of therapy, those considerations necessarily await answer to the question, *is the observed condition responsible for the patient's symptoms*, or *is it an anatomical variation of the normal which is unrelated to the clinical manifestations?* This question is particularly difficult to answer in the case of a clinical condition such as transpyloric prolapse of the gastric mucosa. Recent experiences with a number of cases of prolapsed gastric mucosa stimulated our interest in the subject, but left us confused as to the significance of the condition. Review of the recent literature only added to the confusion, since a divergence of opinion is recorded by various authors in the frequent articles which have been published.

Our experience with one case in particular has impressed us with the fact that prolapsed gastric mucosa may occasionally provide a suitable explanation for unusual and otherwise unexplained upper abdominal pain and associated gastrointestinal symptoms. We are, therefore, recording this case in detail, together with a brief general review of the subject, and the informative opinions which we have collected in personal discussions and correspondence with internists and surgeons in medical centers in various parts of the country. The latter have been particularly helpful and have enabled us to better crystallize our own opinions regarding the incidence and significance of this condition.

Incidence

Prolapsed gastric mucosa is most often encountered in patients in the fourth decade of life, but has been recorded in persons ranging in age from 20 to 80 years. Most authors report a predilection for the male.

In our personal experience we have collected 9 cases in which prolapse of the gastric mucosa was considered significant. This represents an incidence of 3 per cent since 300 roentgenologic examinations of the upper gastrointestinal tract were performed during this 7 months period. Mild or moderate prolapse as indicated by minimal roentgenologic findings was not recorded, especially when it was associated with other pathological conditions which served as a better explanation for the patient's symptoms.

Etiology

The etiology of this condition is somewhat obscure, partly because the pathologic material obtained from resected specimens fails to show consistent findings

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in all cases. Several theories have been advanced. Eliason, Pendergrass and Wright[6] believe that a low grade inflammation of the antral mucosa causes a hypertrophy of the mucosa and that the added effect of hyperperistalsis forces the gastric mucosa through the pylorus. Rees[15] suggested that a degeneration of the pyloric muscle occurs and that as hyperperistalsis develops this causes the antral mucosa to become loosened and forced through the pylorus. Bralow, et al[2], theorize that a hypertrophic gastritis develops secondary to a benign peptic ulcer on either the gastric or duodenal side of the pylorus and in a small number of cases it eventually causes the prolapse of the mucosa through the pylorus. Scott's[19] observations led him to believe that a pre-existing disease process of the gastric mucosa is not necessary for the production of prolapse. He believes that the development of prolapsed mucosa is inherent in the walls of the normal stomach and that any condition which produces hyperperistalsis, whether neurogenic, or caused by chemical stimulation, such as coffee, tobacco, alcohol and condiments, may play a role in the production of the condition. Cove and Curphey[4] offer no new theory but conclude that the lesion is fundamentally a mechanical one based on hypermotility of the gastric mucosa. This hypermotility may be congenital or it may be caused by conditions that produce abnormal gastric motility which, over a period of time, loosens the mucosa from the submucosa to such an extent that prolapse is possible.

There is a common denominator occurring in most theories of the etiology of prolapsed gastric mucosa. This is a disturbance of gastric motility and peristalsis. The most common continuous and effective means for altering gastric function springs from the emotions and the nervous system. Consequently, it would appear that certain neurogenic factors are the inciting causes of the disturbed gastric function and motility which may ultimately result in prolapse of the gastric mucosa into the duodenum. Although it is believed that a pre-existing disease process may not always be necessary for the production of prolapsed gastric mucosa, the condition is most often found in conjunction with either peptic ulceration, hypertrophic gastritis, or both.

Pathology

The pathology involved in prolapse of the gastric mucosa through the pylorus is a redundancy of the mucosa of the pyloric portion of the stomach, often with hypertrophy of the gastric rugae. At operation the prolapsed portion appears like a loose collar of redundant hypertrophied mucosa. The mucosa is not only redundant but its mobility on the submucosa and the muscularis is greater than normal. The pyloric muscle may show hypertrophy. Microscopic examination of the gastric mucosa in the prepyloric area, or in the prolapsed portion, usually reveals hyperemia without other evidence of inflammatory change. Some observers have recorded the presence of inflammatory infiltration into the mucosa while others have found a narrowing of the pylorus with loss muscular of substance.

Symptomatology

The symptoms resulting from prolapse of the gastric mucosa into the duodenum are usually those of the diseases with which the condition is associated, most

often peptic ulcer. There is reason to believe that when prolapsed gastric mucosa complicates a more specific clinical entity, such as peptic ulcer, the usual symptoms of that entity may be altered or obscured to some degree; or that the superadded prolapse may at times be a factor in the production of a complication such as pyloric obstruction or hemorrhage or significant proportions.

In the unusual instance in which prolapsed gastric mucosa occurs without concomitant or associated disease in the upper gastrointestinal tract, symptoms can be expected to be vague and ill-defined, and may include upper abdominal pain, gaseous indigestion, a tendency to regurgitate sour gastric contents, nausea, and occasionally vomiting. Certainly there is no characteristic symptom complex associated with prolapsed gastric mucosa which will permit definite clinical recognition of its presence.

Whether or not prolapsed gastric mucosa alone is capable of resulting in gastrointestinal bleeding of significant proportions remains a question to be settled. It is believed by many authors to be a relatively common cause of hematemesis or melena, or both, and has been so reported[9]. We believe that it is not unreasonable to implicate prolapsed gastric mucosa in the etiology of otherwise unexplained upper gastrointestinal bleeding, although we have not observed massive bleeding from this condition. It is easy to imagine that the traumatized and constricted segment of mucosa would suffer an impaired and disturbed circulation and that significant erosions could be expected to develop in the anoxic tissue. The circumstances here are analogous to the traumatizing effect of the diaphragm constricting the herniated portion of the stomach in a hernia through the esophageal hiatus[11].

The physical findings in most cases are entirely noncontributory. The majority of our patients were found to have localized epigastric tenderness. Studies of gastric physiology and secretion by fractional gastric analysis yield variable findings which cannot be correlated in such a way as to provide an aid in diagnosis.

DIAGNOSIS

Although prolapse of the gastric mucosa into the duodenum is an anatomical condition which can be positively identified only at surgical exploration or postmortem examination, it is possible to recognize the condition without difficulty on roentgenologic study in most cases. It's roentgenologic characteristics are distinctive, and have been adequately summarized and demonstrated by Feldman[8]. The preoperative diagnosis of prolapsed gastric mucosa is made almost exclusively on the basis of these roentgenologic findings.

The most constant and characteristic roentgenologic feature of prolapsed gastric mucosa is the presence of an umbrella-like or mushroom-shaped filling defect in the base of the duodenal bulb, which is usually best demonstrated with the patient in the prone position. The appearance of the duodenal bulb is variable, and the defect may change in extent or it may disappear on different examinations or during different parts of the same examination (figs. 1, 2). Gastric peristalsis is usually found to be hyperactive, and the gastric mucosal markings are often irregular and the folds are increased in caliber. The pylorus

FIG. 1. Spot roentgenogram demonstrating normal duodenal bulb in the upright position

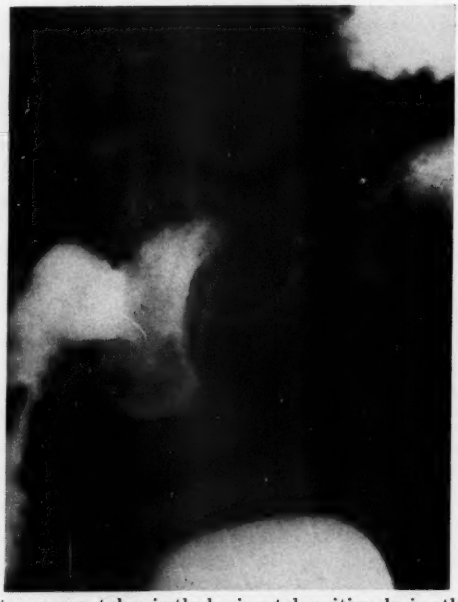

FIG. 2. Spot roentgenogram taken in the horizontal position during the same examination as in figure 1, demonstrating moderate prolapse of the gastric mucosa.

may be widened or it may be narrowed. In many instances the multiple linear filling defects in the pylorus which represent the gastric rugae can be traced into the defect in the base of the duodenal bulb. Some degree of pyloric obstruction may be demonstrated. In all cases, the recognition of the presence of prolapsed gastric mucosa should stimulate the examiner to make an especially careful search for the presence of a peptic ulcer or some other more specific and common clinical entity. This is especially true when marked pyloric obstruction is present. Care should be taken to ecxlude the presence of polyps in the duodenal bulb, and prolapse must be differentiated from neoplastic disease, duodenitis, and other conditions.

Scott[19] believes that prolapsed gastric mucosa is commonly overlooked for three reasons: 1, the examiner is not thinking of the condition; 2, the filling defects produced by prolapse are confused with ulcer or duodenitis; 3, the examiner may fail to report the condition because he attaches no clinical significance to it.

TREATMENT

When prolapsed mucosa is associated with hypertrophic gastritis of significant degree, a peptic ulcer, or some other specific clinical entity, treatment should be directed primarily toward the latter conditions in the usual fashion, with only secondary consideration being devoted to the prolapse. Minor degrees of prolapse, as indicated by minimal roentgenologic findings, are probably best disregarded.

When marked prolapse is found without complicating or associated disease, the patient should be given a prolonged and adequate trial on medical therapy, utilizing the various antispasmodics and gastric sedatives, together with a bland diet and other measures designed to inhibit gastric motility. A majority of patients will respond satisfactorily to such a program, and it is probable that patients continuing to have severe symptoms after an adequate period of such management will be rarely found. In the presence of complications, such as obstruction or hemorrhage, or if the patient continues to have severe and disabling symptoms, and other disease conditions have been carefully excluded, surgical intervention should be considered.

Several types of operations have been suggested and performed. These include; 1) excision of the redundant segment of mucosa with or without pyloroplasty; 2) gastric resection, or 3) gastroenterostomy. Although our personal experience has been restricted to the use of a limited gastric resection, which includes the pylorus and gastric antrum, we feel that vagotomy may prove to have a place in the treatment of this condition. This would appear reasonable inasmuch as hyperperistalsis is the common denominator in all the suggested theories of etiology, and hypertrophic gastritis is frequently described in the pathologic specimens removed. Vagotomy is undoubtedly a most effective means of limiting gastric motility, and Dragstedt[5] has had excellent results with this procedure in the treatment of hypertrophic gastritis, associated with hypersecretion. Of the surgical procedures directed at the lesion per se, it seems to us that gastric resection, using the Bilroth I type of anastamosis, is the most logical. It appears

unlikely that local excision of the redundant segment of prolapsed mucosa would prevent future prolapse of the remaining prepyloric mucosa, since nothing has been done to remove or alter the etiologic factors. A local excision of the redundant segment accompanied by vagotomy would seem more likely to obviate a recurrence of the condition. It should be remembered that the prolapse may be reduced into the stomach with induction of anesthesia, due to relaxation of the pyloric ring, so that the prolapse may seem not to exist in some cases when search is made for it surgical exploration.

MATERIAL

Our experience includes 9 cases in which prolapsed gastric mucosa was considered to be a factor contributing to the patient's symptoms. Of these, prolapse was accompanied by other pathologic conditions in 6 cases: 3 had a peptic ulcer in the duodenal cap, 1 had a postbulbar duodenal ulcer, 1 had a gastric ulcer, and 1 had severe hypertrophic gastritis as demonstrated by gastroscopic examination. In each case, however, the prolapse was sufficiently pronounced to be considered significant, and in the patients with duodenal ulcer atypical symptoms persisted even when the ulcer was demonstrated to have healed. The gastric ulcer was accompanied by almost complete pyloric obstruction, and was demonstrated at operation to be accompanied by a marked prolapse which appeared to be contributing materially to the obstruction. Two patients with prolapse which was not accompanied by other pathology have responded well to medical therapy. The remaining patient had severe symptoms which failed to respond to intensive medical treatment and was operated upon by one of us (G. F. M.). This case will be reported in detail.

Case No. 1. R. D. This 37 year old white man gave a history of severe, intermittent epigastric pain of about 8 years' duration. The pain was described as sharp, occasionally cramping, and usually brief in duration. His distress usually occurred in episodes four or five times yearly, each episode lasting one to three weeks. Remission between these episodes was not complete, however, since he had some vague gastrointestinal discomfort most of the time. The pain had no characteristic pattern or tendency to radiate. It was never related to meals and was not relieved by eating or by any of the numerous antacid preparations which he had been given. He frequently experienced nausea and vomiting with his episodes of pain, but had not experienced hematemesis or melena.

The patient had been operated upon in 1946 for what was thought to be a perforated ulcer, but he was told that nothing definite was found. He had repeatedly been told (without roentgenologic confirmation) that he had a peptic ulcer, and had been placed on strict programs of medical management for this condition without ever having obtained significant relief of his symptoms. He usually experienced some temporary relief from the administration of sedative and antispasmodic preparations. Roentgenologic examination of the upper gastrointestinal tract had been done on several occasions and no definite ulcer identified. On at least two occasions following study by competent clinicians he was told that he definitely did not have a peptic ulcer.

When first seen by one of us (F. M. S.) on Dec. 4, 1951 he gave a history of severe and constant epigastric pain for at least six weeks, during which time he had been treated with bland diet, hourly feedings of milk and cream, alternating with an antacid powder, and methantheline bromide in a dosage of 100 mg. every six hours. This program afforded him no relief whatsoever.

FIG. 3. Case 1. Roentgenogram demonstrating marked transpyloric prolapse of the gastric mucosa.

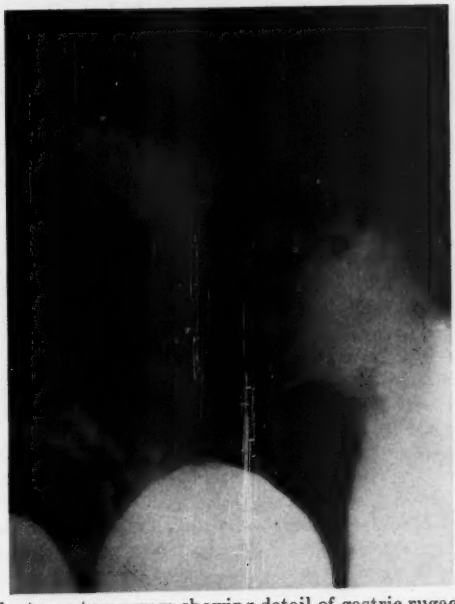

FIG. 4. Case 1. Spot roentgenogram showing detail of gastric rugae in pylorus and base of the duodenal bulb.

The general physical examination was entirely noncontributory, except for the presence of mild epigastric tenderness. The routine laboratory studies and several tests of liver function were well within normal limits. The stools were negative for occult blood. Gastric analysis was done and the fasting specimen contained 21 units of free HCl with 38 units total. The patient was unable to tolerate the tube and additional specimens for the fractional analysis could not be obtained. Gastroscopic examination was refused.

Roentgenologic examination showed the esophagus and stomach to be normal, but revealed a characteristic umbrella-shaped or mushroom-like defect in the base of the duodenal bulb, into which the gastric rugae could be traced (fig. 3, 4). At fluoroscopic examination there was some hestiation in the passage of barium through the pylorus and it was necessary to wait approximately 20 minutes before the duodenal cap could be filled and identified. However, barium subsequently appeared to pass without difficulty through the pylorus and there was no significant gastric retention. The presence of prolapsed gastric mucosa was suspected during the fluoroscopic examination and attempts to force the prolapsed segment back into the stomach were unsuccessful. The second and third portions of the duodenum were normal. No ulcer crater could be identified, and the duodenal bulb was not otherwise deformed.

Operation was done by G. F. M. on Dec. 17, 1951. On opening the peritoneal cavity there were a few adhesions around the gallbladder and duodenum which were attributed to previous surgery. Careful dissection down to the duodenum failed to reveal any evidence of a scar in any part of the duodenum. The gallbladder, common duct, and liver appeared entirely normal. The stomach was opened longitudinally approximately 1 inch proximal to the pyrloric ring, and then opened down to the pylorus. Edematous gastric mucosa protruded through and almost occluded the pyloric ring. A small curved clamp was passed through the pylorus and the pyloric ring was opened, and a specimen for biopsy was taken from the prolapsed segment of mucosa. This was done in order to avoid trauma which might distort the picture during the resection which was to be carried out. In addition a specimen for biopsy was taken from the gastric mucosa in the body of the stomach for comparison. Careful palpation and inspection of all parts, including the interior of the stomach and duodenum, revealed no evidence of ulceration or neoplasm. No trace of a previously existing peptic ulcer or of a previously perforated duodenal ulcer could be seen. A posterior Polya type of gastric resection was done, removing approximately 25 per cent of the stomach.

Pathologic Report

The resected portion of the stomach grossly showed a moderately hyperplastic mucosa. This hyperplastic gastric mucosa protruded through the pyloric ring and appeared hyperemic. Microscopically, there was evidence of increased vascularlty of both the mucosa and submucosa, together with infiltration of inflammatory cells (lymphocytes, with some eosinophils) between the mucosal glands and extending down into the submucosa. Some lymphoid hyperplasia was present, the follicles being nicely formed. The usual gland cells and structures were recognized, and all were uniform and orderly. The greatest extent of inflammation and vascularity was present in the prolapsed segment of the pyloric mucosa. These changes became progressively less toward the proximal portion of the resected specimen, where there was only slight evidence of superficial inflammation. No ulcerations were present in the pathologic material.

The patient has remained free of all symptoms, including pain, since leaving the hospital. When contacted in October 1952, 10 months after his operation, he stated that he was feeling well and having no distress referable to the gastrointestinal tract. Prior to operation, he had not been entirely free of symptoms for more than a few weeks for a period of eight years. Progress roentgenologic examination of the upper gastrointestinal tract, six weeks after his operation, revealed a normally functioning gastroenterostomy without evidence of obstruction or ulceration.

DISCUSSION

Some degree of prolapsed gastric mucosa is undoubtedly present in a significant percentage of patients undergoing roentgenologic study of the stomach and duodenum. How much prolapse is necessary to cause symptoms is open to question. Bockus[1] believes that only severe prolapse associated with gastritis can cause symptoms and then only after producing partial pyloric obstruction.

Numerous authors[8, 17, 18], have emphasized the danger of overlooking some associated pathology, such as peptic ulcer, neoplasm, or gastritis as the lesion responsible for the symptoms. As the case reports are studied one is impressed by the ulcer-like syndrome which is present in most of the cases. Ruffin[18] emphasizes that failure to demonstrate an ulcer on roentgenologic examination does not exclude the possibility that one is present. Nor does failure to demonstrate an ulcer in the second portion of the duodenum on the posterior wall at the time of operation exclude the possibility unless the duodenum is opened and explored. Furthermore, resection of the lower portion of the stomach for the prolapse is standard treatment for an ulcer, and relief of symptoms following this procedure does not prove that the symptoms were due to prolapse.

Palmer and Kirsner[13] have never diagnosed prolapsed gastric mucosa as a cause of pyloric obstruction or hemorrhage, nor have they regarded it as a significant cause of clinical symptoms; however, they do not exclude the possibility of severe prolapse causing obstruction and significant clinical symptoms. It was their impression, on the basis of an extensive experience, that this lesion is uncommon and that the majority of instances of prolapsed mucosa are coincident with roentgenologic observation and not responsible for the symptoms leading to the examination.

McHardy[12] believes that mucous membrane prolapse is frequently found but frequently can not be proved to be the significant cause of important clinical manifestations, because of the difficulty in correlation and lack of specificity of symptomatology. He does, however, believe that it is of clinical importance, and that it may occur coincidently with other diseases which are excited by that entity, and that it also may occur independently of other disease, and that it may cause manifestations simulating an ulcer syndrome.

Levy[10] states that prolapsed gastric mucosa could easily be responsible for unexplained gastrointestinal symptoms, including hemorrhage, but is uncertain as to the importance of the amount of prolapse which is frequently seen on roentgenologic study. It was Levy's belief that in all cases with prolapse of the gastric mucosa the associated pylorospasm was the major factor in the production of symptoms.

Collins[3] tells us that although the condition is occasionally recorded in the roentgenologic impression, it is rarely, if ever, utilized as an explanation for significant gastrointestinal symptoms in his department. He is unable to recall, after conversations with a number of his colleagues, a single case which had been operated upon at the Cleveland Clinic for this condition or where this condition was found at operation when exploration was done for other entities. Patients at

the Cleveland Clinic reported at roentgenologic examination to have prolapsed gastric mucosa are always followed carefully with progress roentgenologic study. Collins has noted that the roentgenologic appearance is rarely, if ever, changed even though the patient is completely relieved of his symptoms. Some of these patients have been followed over a period of at least 10 years.

Patterson[14] believes that prolapse of the gastric mucosa is commonly associated with a peptic ulcer syndrome, usually duodenal ulcer. Waugh[20] says that it is occasionally seen by roentgenologists at the Mayo Clinic but that it is the opinion of the clinicians that it is only the very special case in which it is responsible for symptoms. He has not had occasion to operate upon a patient when this diagnosis was made. He states that he would be very hesitant to ascribe any symptoms to this finding. Dragstedt states that he has rarely had occasion to deal with the problem of prolapse of the gastric mucosa. Renshaw[16] believes that prolapsed mucosa may occasionally cause significant upper gastrointestinal symptoms. He has not had occasion to resort to surgery in the treatment of prolapsed mucosa. Feldman[7], who has made an exhaustive study of the subject, believes that prolapse of the gastric mucosa is a definite clinical entity, and that a definite clinical pattern can be recognized, especially in the more severe cases. It is his opinion that its incidence is much higher than has been indicated in studies reported to date. He says that symptomatology severe enough to warrant operation may be found.

SUMMARY

From this study we conclude that a cautious and conservative attitude is indicated in evaluating prolapsed gastric mucosa, for it is known that the demonstration of an abnormality does not prove casual relationship to the symptoms experienced by the patient. Probably the initial reaction of the clinician, on recognizing that some degree of prolapse of the mucosa is present, should be to look with special care for a peptic ulcer, a prolapsing polyp, antral gastritis, hiatal hernia, or some other associated pathology.

It is unreasonable to deny the existence of prolapse of the gastric mucosa into the duodenum, for some degree of prolapse is present in a considerable percentage of patients having roentgenologic examinations of the upper gastrointestinal tract, and the condition has been anatomically demonstrated at operation in many recorded instances. Our personal experience, including the case cited, has fortified our impression that prolapse of the gastric mucosa into the duodenum is an entity which may be rarely but definitely responsible for significant and otherwise unexplained gastrointestinal symptoms. It is probable that it is capable of producing significant gastrointestinal bleeding or partial pyloric obstruction on rare occasions. When roentgenologic study indicates that a marked degree of prolapse is present, the finding should not be ignored, nor should great significance be attributed to it, especially until other pathologic conditions have been carefully excluded. The finding must be carefully evaluated in the light of all the clinical, laboratory, and roentgenologic findings.

In those individuals who have an ulcer-like syndrome the usual therapy for

peptic ulcer should be instituted. When the patient's symptoms are not suggestive of peptic ulcer, and ulceration can not be demonstrated by appropriate studies, he should be treated with a bland diet, avoiding factors known to stimulate gastric peristalsis, and antispasmodic preparations. Ganglionic blocking agents, such as methantheline bromide, are probably the most effective in the reduction of gastric motility, and should therefore provide maximum relief.

Surgical treatment should be reserved for that group of patients in whom prolapse is complicated by bleeding, pyloric obstructions or severe pain which fails to respond to careful and prolonged medical management. It appears unlikely that careful surgeons will find prolapsed gastric mucosa to be a frequent reason for surgical intervention.

REFERENCES

1. Bockus, H. L.: Gastroenterology, Vol. 1, 762, Philadelphia, W. B. Saunders Co., 1943.
2. Bralow, S. P., Melamed, M., and Hiller, R. I.: Prolapsed gastric mucosa; roentgenologic demonstration of ulcer crater in prolapsed polypoid mucosa, Am. J. Digest Dis. 10:93, 1943.
3. Collins, E. N.: Chairman of section on Gastrointestinal Diseases, The Cleveland Clinic, personal communication.
4. Cove, A. M., and Curphey, W. C.: Prolapse of redundant gastric mucosa, Surg., Gynec. & Obst. *88:* 108 (Jan.) 1949.
5. Dragstedt, L. R.: Professor and chairman, Department of Surgery, University of Chicago, personal communication.
6. Eliason, E. L., Pendergrass, E. P., and Wright, V. W. M.: Roentgen-ray diagnosis of pedunculated growths and gastric mucosa prolapsing through pylorus, Am. J. Roentgenol. *15:* 295 (April) 1926.
7. Feldman, M., Morrison, S., and Myers, P.: The clinical evaluation of prolapse of the gastric mucosa into duodenum, Gastroenterology *22:* 80 (Sept.) 1952.
8. Feldman, M., and Myers, P.: Roentgen diagnosis of prolapse of gastric mucosa into duodenum, Gastroenterology *20* No. 1: 90 (Jan.) 1952.
9. Kaplan, I. W., and Shepard, R. M.: Prolapse of gastric mucosa into the duodenum, J. A. M. A. *147* No. 6: 554 (Oct. 0) 1951.
10. Levy, J. S.: Professor of Medicine and Gastroenterology, University of Arkansas, personal communication.
11. Madding, G. F., and Spencer, F. M.: Esophageal hiatus hernia in older persons, Texas State J. Med. *47* No. 11: 748 (Nov.) 1951.
12. McHardy, G.: Assistant Professor of Medicine, Tulane Medical School, personal communication.
13. Palmer, W. L.: Professor of Medicine, and Kirsner, J. B.: Associate Professor Clinical Medicine, University of Chicago, personal communication.
14. Patterson, C. O.: Clinical Associate Professor of Medicine and Gastroenterology, Southwestern Medical School, personal communication.
15. Rees, C. E.: Prolapse of gastric mucosa through pylorus; surgical treatment, Surg., Gynec. & Obst. *64:* 689 (March) 1937.
16. Renshaw, R. J. F.: Internist, Santa Ana, California, personal communication.
17. Rubin, J. S.: Prolapse of polypoid gastric mucosa into the duodenum with malignant change, Radiology *38:* 362 (March) 1942.
18. Ruffin, J. M.: Professor of Medicine, Duke University School of Medicine, personal communication.
19. Scott, W. G.: Radiographic diagnosis of prolapsed redundant gastric mucosa into duodenum, with remarks on clinical significance and treatment, Radiology *46:* (June) 1946.
20. Waugh, J.: Associate Professor of Surgery, University of Minnesota Medical School, Mayo Clinic, personal communication.

FEMORAL ARTERIOVENOUS FISTULA WITH FALSE ANEURYSM

REPORT OF A CASE WITH EARLY RESTORATIVE OPERATION

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Arteriovenous fistulas are commonly found in military surgical practice and hundreds of cases have been treated in the army vascular centers.[1, 2] The lesion will also be found in civilians who have had gunshot or stabbing injuries. Surgical

FIG. 1. Drawing showing relation of hematoma (false aneurysm) to femoral vessels. and the preliminary incision to expose the femoral artery (arrow).

management has not become completely standardized, since a waiting period of at least three months for the development of collateral circulation has been commonly advised and excision of the fistula with quadruple ligation of the involved artery and vein has been considered to be acceptable, even though the

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functional results have not been good in many instances. This case report is illustrative of the fact that when modern methods of vascular repair are utilized, it is possible to carry out very early definitive measures with gratifying results.

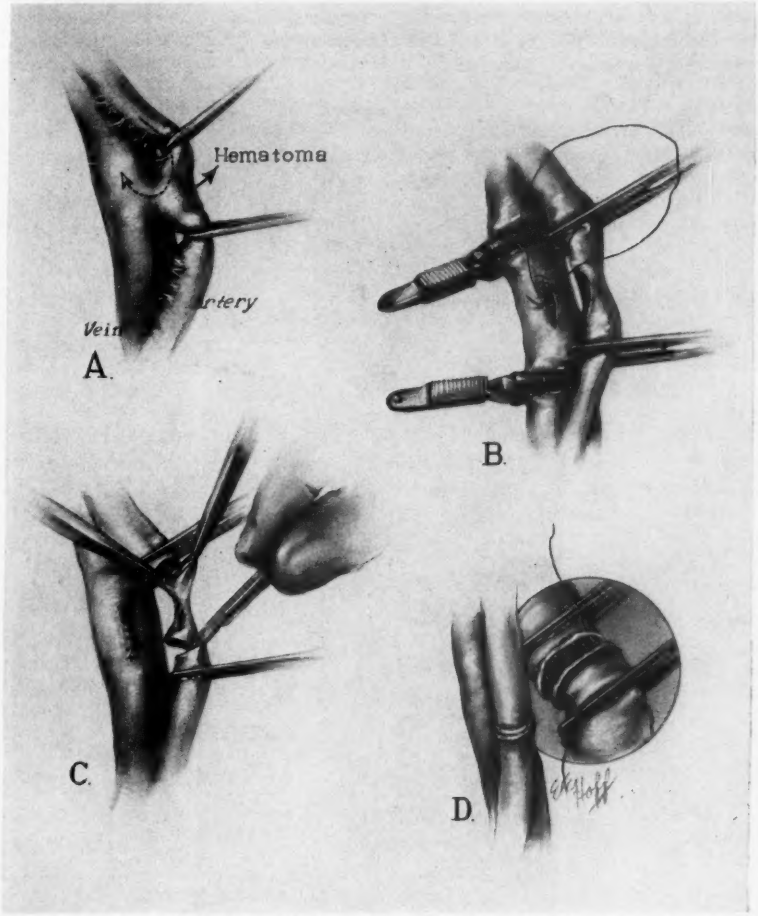

FIG. 2. Steps in the repair of the femoral arteriovenous fistula with associated false aneurysm.

Case Report. V. F., a 37 year old white man, was admitted to the Henry Ford Hospital on March 10, 1951. Five weeks before, while working at his trade as a butcher, he received an accidental stab wound in the middle of the left thigh. There was brisk hemorrhage with spurting of blood which was eventually controlled by the application of pressure. He was taken to another hospital where ambulatory treatment with pressure dressings was continued. A large painful swelling appeared in the anterior portion of the thigh, over which a continuous bruit could be heard. Because of further enlargement of the mass and the possibility of its rupture to the exterior, the patient was referred for surgical treatment.

Examination of the left leg revealed a large, firm ovoid mass in the thigh, the extent of which is indicated in figure 1. The surrounding tissues were ecchymotic. A scar measuring 1 cm. in length marked the point of entrance of the knife. The mass did not pulsate; a continuous thrill was palpable and a very loud bruit was audible through the stethoscope. A pulse was palpable in the posterior tibial artery. The systemic blood pressure was 138/84. A roentgenogram of the chest did not show hypertrophy of the heart. The red blood cell count was erythrocytes 2,890,000 per cu. mm., the hemoglobin was 12 Gm. A transfusion was given to correct the posthemorrhagic anemia.

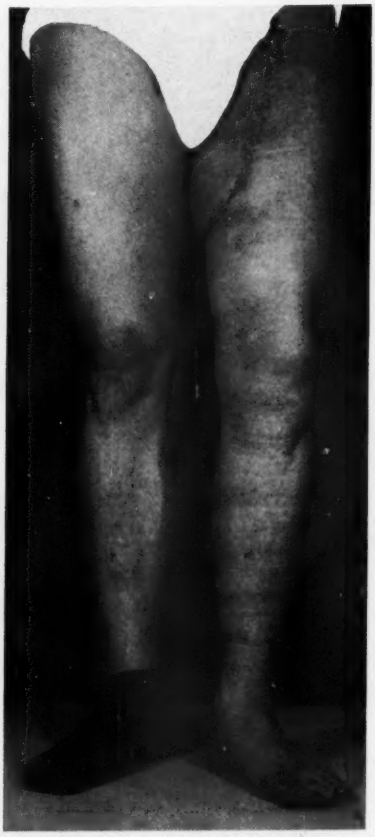

FIG. 3. Photograph of lower extremities of patient one month after operation

Operation for the repair of the arteriovenous fistula with associated large false aneurysm was done on March 13, 1951. First, the femoral artery was exposed immediately below Poupart's ligament and a tape was passed around it (fig. 1). The incision was then continued inferiorly and medially to pass through the scar at the point of entry of the knife (fig. 3). The upper part of the hematoma under the femoral fascia was then entered and there was an immediate escape of clotted blood under pressure. A bulldog clamp was applied to the femoral artery. The hematoma cavity was opened widely and a large quantity of clot was removed. At the bottom of the wound, it was found that blood was escaping from an endo-

thelial-lined opening which was first thought to be the femoral vein but was subsequently shown to be the opening from the artery into the false aneurysm (fig. 2 A). This opening was closed with the finger until the vein and artery were mobilized above and below the lesion so that occluding clamps could be placed. The second opening in the femoral artery connecting with one in the vein could be demonstrated. The two vessels were separated, and the hole in the vein was closed with a longitudinal running suture of No. 00000 Deknatel silk (fig. 2 B). The two openings in the femoral artery were closed in a similar manner, but the resulting constriction did not permit enough blood to flow into the distal portion to produce a pulsation. This part of the artery was excised (fig. 2 C) and after further mobilization of both the proximal and distal segments, it was possible to make an end to end anastomosis. This procedure was greatly facilitated by the use of the Potts serrated ductus clamps, which permitted considerable traction to be placed on the arterial segments without slipping or damage to the vessel wall (fig. 2 D). Following the removal of the clamps from the femoral artery, there was excellent pulsation in the distal portion of the vessel. After thorough irrigation and the instillation of penicillin, the wound was closed and drained. Anticoagulants were not used.

The postoperative course was satisfactory. A good posterior tibial pulse was always palpable. Ambulation was permitted on the fifth day. Belause of a slight tendency toward swelling, an elastic bandage was worn below the knee for a short time (fig. 3). He returned to work six weeks after the operation. A report from the patient a year later stated that there was no disability with regard to the left leg. Slight swelling of the ankle was noted after long periods of standing.

Comment. The advantages of the re-establishment of the continuity of the major vessels in the treatment of arteriovenous fistula are obvious. Excellent function of the involved part is to be expected, and the definitive operation can be carried out as soon as the diagnosis is made.

REFERENCES

1. Freeman, N. E.: Arterial repair in the treatment of aneurysms and arteriovenous fistulae; report of 18 successful restorations, Ann. Surg. *124:* 888 (Nov.) 1946.
2. Seeley, S. F., et al: Traumatic arteriovenous fistulas and aneurysms in war wounded; a study of 101 cases, Am. J. Surg. *83:* 471 (March) 1952.

ACUTE PSEUDOMEMBRANOUS ENTEROCOLITIS SIMULATING ACUTE SURGICAL DISEASES OF THE ABDOMEN

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Acute pseudomembranous lesions involving the mucosa and submucosa of the gastrointestinal tract, and varying in their location from the esophagus to the rectum, have been described as occurring in association with a wide variety of diseases. Bockus[1] has stated that a membranous type of enteritis may occur in pyremia or septicemias of various types. Any of the infectious diseases are capable of causing severe inflammatory changes in the stomach and intestines. Notable examples are typhoid fever, the dysentery group, enteric tuberculosis and cholera. Acute enteritis of varying severity may develop as an extension of a neighboring process, such as peritonitis or abdominal carcinoma, and invariably occurs in conjunction with chronic intestinal obstruction. Diphtheria very rarely is associated with a membranous type of enteritis. Poisoning from arsenic, lead, mercury and other inorganic toxic substances may give rise to very acute inflammatory changes in the small intestine.

The inflammatory changes which occur in the lower end of the ileum and proximal colon in association with infectious diseases and with acute toxic poisoning are thought to be due to the excretion of toxic materials from the blood stream through the intestinal wall. In the terminal stages of some serious constitutional conditions, such as malignant disease, anemia, Addison's disease, severe burns and nephritis with uremia, extensive destruction with sloughing of the intestinal mucosa is sometimes found. The pathogenesis of these intestinal lesions is not clearly understood. Experimentally, extensive inflammatory intestinal lesions have been produced by merely injecting intravenously a solution of 10 per cent urea.

That acute pseudomembranous enterocolitis may occur as a complication following surgery has been known since Finney[3] in 1893 described a patient on whom he had performed a gastroenterostomy, but who died on the fifteenth postoperative day of severe "diphtheritic" colitis. Penner and Bernheim[7], in a review of cases encountered at the Mount Sinai Hospital in New York, called attention to the pathologic aspects of the lesions and postulated that shock might be an important factor in the development of the membranous lesions. They pointed out that in shock the venules and capillaries of the intestinal mucosa are engorged with blood and, in the early stages, there is simple edema of the mucosa and submucosa. Later there are pericapillary hemorrhages and finally focal necrosis of the mucosa which, by fusion and extension, may give rise to the late pseudomembranous lesions.[8] Dixon and Weismann[2] reviewed the records at the Mayo Clinic from 1940 to 1947 and found 23 cases of acute pseudo-

* From the Ferrell-Duncan Surgical Clinic. Presented during the annual assembly of The Southwestern Surgical Congress in Dallas, Texas, October 20–22, 1952.

membranous enterocolitis occurring as a postoperative complication. Twenty-one of these 23 cases were verified at autopsy and 2 cases presumed to have had the disease recovered under intensive treatment.

The disease occurred from the fourth to thirty-sixth postoperative day but the majority of cases were found during the first two weeks following operation. The cases were equally distributed between the two sexes and occurred following a wide variety of operations, but seemed to be most common after procedures on the gastrointestinal tract. Although they could assign no valid reason, the incidence of the disease led them to believe that it is apparently on the increase. No definite etiologic factor could be cited as a primary cause.

Peritonitis was present in a few of the cases and when it is present the authors suggested that the toxic character of the infection and that the severe debility of the patient are important factors in the predisposition of the intestine to development of membranous lesions. They could not assign a definite role to infection as the etiologic factor. In most of the cases the patients had been hospitalized preoperatively and had been given chemotherapeutic agents to prepare the bowel for operation. Cultures of fecal discharges were negative for the usual bacillary or parasitic agents which produce primary enteritis. Secondary infection of an already denuded bowel would be expected and was indicated by the marked purulent character of the fecal discharge which occurred late in the disease.

The most striking manifestation of the disease is that of severe progressive circulatory collapse which is extremely resistant to supportive measures, and which results in irreversible shock and death in a matter of hours.[2, 4, 6] Dixon states that the similarity to Asiatic cholera has been noted and postulated that the clinical manifestations may be produced on a similar basis to that of cholera. The interval between the state of shock and death was so brief in many of his cases that he believed it unlikely that extensive pseudomembranous lesions could have developed subsequent to the state of collapse.

Diarrhea in the series of cases reviewed by Dixon and Weismann was present in only 9 of the 23 cases. In some of these cases the stool was described as "rice water" in character and in the 2 surviving cases the fecal discharge became progressively more foul and purulent and contained nondescript shreds or plaques of material thought to be sloughed tissue. However, most of the cases did not show pronounced increase of intestinal activity or marked loss of fluid externally. Bleeding from the bowel was not noted. In the cases in which there was no significant diarrhea it was thought probable that the loss of intracellular and extracellular fluid into the lumen of the intestine would partially account for the circulatory collapse.

Mild abdominal cramps and moderate distention of the paralytic type was present in the majority of the patients and usually preceded circulatory collapse by only a few hours. Dilatation of the bowel was usually progressive after onset of shock. Nausea and vomiting were present in some degree, but usually not pronounced. Dark brown material, suggestive of old blood, was vomited or aspirated from those cases that had upper gastrointestinal tract involvement. Fever, to any marked degree, was a terminal event and was thought to be due to

extensive secondary infection of the intestinal wall with absorption of toxic products.

Kleckner et al[6] of the Mayo Clinic made a clinicopathologic study of 14 cases in which the disease was not preceded by an operation. In 7 of the 14 cases the pseudomembranous enterocolitis was considered the immediate cause of death. In the remaining cases only small segments of the intestine were involved and it could not be considered the chief cause of death. Associated pathologic conditions were present in all of the 7 cases and included 4 cases of obstruction of the large bowel from carcinoma; 2 cases of cardiac disease; and 1 case of streptococcic septicemia and bronchopneumonia. The clinical features of these cases were very similar to those reported by Dixon. They state that because of the explosive onset of the disease and rapid down-hill course ending in death, laboratory tests were of little aid in the diagnosis. The most significant laboratory findings were leukocytosis, increased sedimentation rate and hemoconcentration. Azotemia occurred in a few cases. The most significant roentgenologic finding was distention of the intestine of a paralytic type. At auotpsy large portions of the small intestine and colon were covered by the typical diphtheritic type of membrane. They were unable to conclude that the membrane was formed after onset of shock in the cases.

Positive diagnosis of the disease apparently can be established only at autopsy or by identification of pseudomembranous casts in the fecal discharge.[2, 5, 9] However, the diagnosis should be suspected in a patient who presents the clinical picture just described and in whom other intra-abdominal pathology can be excluded. The prompt recognition and institution of intensive fluid replacement therapy constitutes the only valid hope of saving the patient. Dixon recommends the intravenous injection of 1000 to 2000 cc. of whole blood and additional plasma up to 3000 cc., depending upon the response of the circulatory system. In addition, depending upon loss from fecal discharge or gastric suction, he gives up to 4000 cc. of glucose in water or sodium chloride solution.

Our interest in the disease was stimulated by observing a case in which the onset of collapse was accompanied by abdominal findings of such nature that it was thought we were confronted with an acute surgical emergency.

CASE REPORT

The case is that of a 49 year old woman who entered the hospital because of two episodes of mild abdominal cramping and loose stools numbering from 2 to 4 per 24 hours. Two weeks prior to admission she had had an episode of mild diarrhea and cramping and because her husband was also having the same trouble she had taken a course of medicine which consisted of 2 Gm. of succinyl sulfathizaole four times daily. The symptoms abated and she was up and around, carrying on her usual activities. One week later she had a second episode of diarrhea and mild cramping, but this time she was given aureomycin, 250 mg. every six hours. Again the symptoms cleared up, only to recur two days before entering the hospital. She stated that she was only moderately ill; had been eating most of her meals regularly; was up and about during the day and slept fairly well at night. Her main complaints were a feeling of generalized malaise; at times a feeling of slight fever and intermittent abdominal cramping followed at times by semi-liquid stools.

Review of the systems was noncontributory. Her menses had stopped one and a half years before and there had been no difficulty since then. Her past history revealed she had

had only the usual childhood diseases. Twenty-three years ago she had a pelvic operation which consisted of removal of the right ovary, both tubes and a perineal repair. She was married and had borne four children.

Physical examination revealed a well developed and well nourished woman of about her stated age of 49 years. She did not appear acutely ill. She weighed 165 pounds. The blood pressure was 104/68, pulse was 82 and her temperature was 99.2 F. Examination of the head, neck and chest was entirely negative. Her abdomen was soft and not distended. There was slight tenderness to pressure in the lower abdomen, but no rigidity or rebound tenderness. There were no palpable masses. Pelvic and digital rectal examinations were negative.

Laboratory studies revealed a hemoglobin of 11.5 Gm. or 66 per cent of normal; erythrocytes 3,640,000 per cu. mm. and leukocytes 13,600 per cu. mm. with 70 per cent polymorphonuclear cells. Urinalysis was normal. Stool examination was negative for pus, blood or intestinal parasites.

Investigation of the intestinal tract by roentgenogram was planned for the following day, but was postponed because the patient's discomfort had increased. She was slightly nauseated and complained of intensified abdominal cramps. However, she had only one soft bowel movement during the first 24 hours after admission to the hospital. Her abdomen was essentially the same as on admission, except for an increase in peristaltic activity. Her afternoon temperature rose to 100.2 F. with a pulse rate of 100.

She was given 400,000 units of penicillin and 1 Gm. of streptomycin intramuscularly and also 2 Gm. of succinyl sulfathiazole by mouth four times daily. Because of her nausea, 1000 cc. of 5 per cent glucose in water was given intravenously.

The following day, two days after admission, her condition was essentially unchanged. She was still somewhat nauseated, but her abdomen was soft and not distended. There was some increase in tenderness over the lower abdomen. Her temperature rose to 100.4 F. in the afternoon and a white blood count was leukocytes 11,000 per cu. mm. with 56 per cent polymorphonuclear cells. Late that afternoon she had a small emesis and three liquid bowel movements.

Early that evening she looked ill for the first time and there was some beginning distention of the abdomen with increased generalized abdominal tenderness. She had a second small emesis at 10:00 p.m. and at 12:00 p.m. the nurse reported her condition had suddenly become much worse. She looked extremely ill, complained of severe nausea and was moaning with pain. Her skin was cold and she was sweating profusely. Her temperature was 98.9 F. and the pulse rate was 128, weak and thready in character. The blood pressure was 60/45. The abdomen was much distended and exhibited a marked generalized rigidity, almost board-like. There was generalized tenderness over the whole abdomen, but most marked over the left lower portion. There was also high grade rebound tenderness over the whole abdomen. The general picture was that of acute abdominal disease with extreme circulatory collapse and shock.

A presumptive diagnosis of perforated viscus with early generalized peritonitis was made, possibly resulting from diverticulitis with perforation. Exploratory laparotomy was elected if the patient's general condition could be improved to a safe level. She was given 1000 cc. of 5 per cent glucose in physiologic sodium chloride solution and 500 cc. of whole blood. Two hours later her blood pressure had risen to 95/60 and her pulse was 112.

The abdomen was opened through a lower midline incision and exploration revealed a greatly dilated, edematous and engorged terminal ileum and colon. It was estimated that there were 300 to 400 cc. of dark straw-colored transudate with many fine particles of fibrin free in the peritoneal cavity. However, careful examination did not reveal any perforation of the intestine. The colon itself was extremely thickened, its walls having a rubbery consistency and the serosal vessels were dilated and engorged. The lumen of the colon contained considerable liquid fecal material.

The patient's general condition was poor during the operation. She received continuous intravenous fluids and whole blood, but a solution of norepinephrine administered by continuous drip was necessary to maintain her blood pressure at a safe level.

Her postoperative course from the time of surgery to her death some 30 hours later was

one of a continuous battle against shock. Treatment consisted of uninterrupted gastric suction, oxygen, antibiotics and intravenous fluids. She received a total of 5500 cc. of fluids, which included 1500 cc. of whole blood and 500 cc. of plasma. The remaining 3500 cc. of fluid consisted of 5 per cent glucose in water and physiologic sodium chloride solution.

About 10 hours postoperatively an electrocardiogram was taken which showed only sinus tachycardia, the rate being 160. There were no findings suggestive of potassium deficiency. At the same time a blood count showed a hemoglobin of 102 per cent with erythrocytes 5,000,000 per cu. mm. The white count was leukocytes 9,300 per cu. mm. with 75 per cent polymorphonuclear cells. The hematocrit reading was 60 per cent. These findings demonstrated a marked hemoconcentration, but during this time she was receiving continuous intravenous fluid through two vena punctures. She was also given 800,000 units of penicillin, 1 Gm. of streptomycin intramuscularly and 1000 mg. of aureomycin intravenously. Twenty cubic centimeters of aqueous adrenal extract was given in the vein and 2 cc. of adrenal cortex in oil intramuscularly every six hours.

Her general condition progressively deteriorated. Her temperature ranged from 100 to 103 F. except terminally, when it rose to 107 F. Her blood pressure was kept above shock level for about 20 hours postoperatively by intermittent administration of norepinephrine solution. During the terminal hours it dropped to 60 systolic and remained at that level regardless of the type of therapy. She excreted 125 cc. of urine during the postoperative period. There was no diarrhea during this period, but intermittent insertion of a rectal tube obtained quantities of dark brown, foul-smelling liquid material. A specimen sent to the laboratory for examination and culture contained some microscopic blood and pus, but no specific organisms were cultured.

At postmortem examination the significant findings were limited to the gastrointestinal tract. The mucosa of the stomach showed a loss of rugae and numerous 1 mm. pits. The small intestine, to a region of about 3 feet from the terminal ileum, was normal. At the region located approximately 3 feet from the ileo-cecal juncture there was a greenish-yellow plastic pseudomembranous exudate which covered the mucosa of the entire terminal ileum and extended throughout the colon. This membrane was quite tenacious and on removal revealed mucosa dusky red in color which showed evidence of edema and congestion. Microscopically, the mucosa of the ileum and colon was markedly disrupted by edema, congestion and a pseudomembrane. The membrane was composed of an inner layer of cellular debris, then a diffuse infiltrated portion of acute and chronic inflammatory cells all embedded in a fibrinous matrix. Only the proximal segments of the glands were present. The submucosa, muscularis and serosa were congested, edematous and mildly infiltrated with plasma cells and mononuclear cells. There was no evidence of fungi or parasites.

REFERENCES

1. Bockus, H. L.: Acute Enteritis in Gastro-Enterology, Vol. 2, 147, Philadelphia, W. B. Saunders Company, 1944.
2. Dixon, C. F., and Weismann, R. E.: Acute pseudomembranous enteritis or enterocolitis; complication following intestinal surgery, S. Clin. North America *28:* 999 (Aug.) 1948.
3. Finney, J. M. T.: Gastro-enterostomy for cicatrizing ulcer of pylorus, Bull. Johns Hopkins Hosp. *4:* 53 (March) 1893.
4. Hilsabeck, J. R., and Dixon, C. F. L: Treatment of acute postoperative pseudomembranous enterocolitis, Am. J. Surg. *84* No. 1: 114 (July) 1952.
5. King, J. W.: Membranous enteritis, Nebraska M. J. *9:* 481 (Dec.) 1924.
6. Klechner, M. S., Bargen, J. A., and Baggenstoss, A. H.: Pseudomembranous enterocolitis; clinicopathologic study of 14 cases in which the disease was not preceded by an operation, Gastroenterology *21:* 21 (June) 1952.
7. Penner, A., and Bernheim, A. I.: Acute postoperative enterocolitis; study of pathologic nature of shock, Arch. Path. *27:* 966 (June) 1939.
8. Penner, A., and Druckerman, L. J.: Enterocolitis as a postoperative complication and its significance, Gastroenterology *11:* 478 (Oct.) 1948.
9. Summerfeldt, P.: Pseudomembranous iliocolitis, Arch. Pediat. *41:* 565 (Aug.) 1924.

TREATMENT OF HERNIAS IN INFANTS AND YOUNG CHILDREN

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The early repair of inguinal hernia in infants and young children has not been widely accepted by pediatricians nor surgeons. A number of articles have been published suggesting trusses, and recommending that the operation be delayed until the infant has attained more maturity. The advice often given to parents is to wait until the child is "older", usually between 4 and 6 years of age. During this time the parents usually worry about incarceration or strangulation, and limit the child's normal activity. In the meantime the hernia often grows larger. Practically all inguinal hernias in this age group are the indirect type. The direct type is so rare that it need not be considered in this discussion.

Inguinal hernias are occasionally discovered at birth or shortly thereafter, but the majority of congenital hernias in infants are discovered during the second and third months of life when the infant is becoming more active. It is at this time that the parents note the mass in the scrotum or inguinal area and consult a physician. Except in malnourished or sickly infants, a repair of the hernia can be safely done at this time. In this age group the dissection of the hernial sac is more difficult than in older children, but with caution, moderate skill and patience, it can be satisfactorily done. There is a great difference between the type of operation needed for a child as compared to the type for an adult. This is an etiologic and anatomic difference which actually makes the earlier repair more certain of cure. An inguinal hernia occurring during infancy or childhood is not the result of muscle or fascial weakness, but due to a failure of the processus vaginalis to become obliterated. During embryological life, before the testes enter the inguinal canal, the processus vaginalis projects down from the peritoneum through the various muscle and fascial planes retaining an opening with the peritoneal cavity. The processus vaginalis is usually obliterated and the opening to the peritoneum is closed. If this does not occur, an open sac remains and crying, coughing or straining may cause herniation of the intestine. A variety of types of reconstructive operations in these young patients have been advocated. Extensive repair, however is not needed. The sac, an embryological remnant, is the single cause of the hernia and its removal is all that is needed for a cure. A very simple operation for hernia repair in the young age group was advocated as far back as 1899 by Ferguson[3] and more recently by Hertzfeld,[4] Coles,[1] and Potts.[5]

The advantages of early operation are many. There is a very low recurrence rate, a low mortality, the wearing of a truss is obviated, there is a much shorter hospital stay which reduces hospital costs and the danger of incarceration with its complications is eliminated. A less known complication, but still an important one, is the injury to the testicles or ovaries, secondary to incarceration. Psycho-

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logically, the hernia patient is protected by early operation. These children often become behaviour problems if the hernia is not repaired early. Their parents have been warned of the complications that can occur and they become over protective, not allowing the child to cry or play normally. With early operation the parents' worries are alleviated concerning the possibility of strangulated hernia. This worry is not without foundation. In Pott's series, 19 per cent of the infants entered the hospital with incarceration or with a history of recent incarceration. A series of similar cases observed between 1945 and 1950 in the Children's Hospital at Denver showed incarceration in 46 per cent of 24 infants under 8 weeks of age and 25 per cent incarceration rate in 105 children of 1 year of age or younger. The possibility of strangulation is precluded by early repair. Strangulation of hernias causes a high mortality rate in any age, but especially in infants. After it is determined that the child has no physical condition contra-indicating operation and the child is found to be developing normally, all of which can be determined, for the most part, at the end of the first month of life, surgical correction of an inguinal hernia may be done.

Inguinal hernioplasty is performed through a transverse incision which will heal with less scar formation if it follows Langer's lines. The incision is made in the skin flexion crease in the lower portion of the abdominal wall over the approximate site of the internal ring. The aponeurosis of the external oblique is split in this area in the direction of its fibers, leaving the fibers of the external crural ring intact. With care and gentleness, the sac is dissected from its adjoining structures down to its neck. The spermatic cord should not be elevated nor traumatized. The neck of the sac is transfixed and ligated with a suture. The dependent portion of the sac is then excised. The separated fibers of the external oblique are approximated; the skin is closed and a small dry dressing, just large enough to cover the wound is applied. The child may leave the hospital the next day providing he is afebrile and is taking a normal diet.

Rarely, an infant may develop a large inguinal hernia with a wide internal opening. When this occurs, a simple reconstructive repair must be done to close the large internal ring. This necessity is the exception to the rule, however, and we have seen but one instance in our series needing this plastic repair where it seemed that the large opening at the internal ring was due to the stretching of the fascia and musculature rather than any attenuation of these structures. A few sutures closing the inguinal ring was the only repair necessary.

Some surgeons have advocated leaving the distal portion of the hernial sac if it extends to, or around the testicle, stating that there is no likelihood of hydrocele formation. No one has reported a follow-up study of this technic through adulthood. It seems possible that the remnant tip of the intrascrotal sac might later develop a hydrocele. Duckett[2] reports 2 cases of hydrocele formation following incomplete removal of a hernial sac in children. We routinely dissect away the entire sac and have seen no evidence of harm as a result. Associated hydroceles in our cases have been routinely removed through the hernia incision.

When a hernia and an undescended testicle coexist, we have deferred surgery until the infant has reached the age of at least 3 years. The cord structures are so small and delicate in infants that any surgical procedure to replace the testicle

in its normal position may cause injury to these structures. We have seen no instance of incarceration in these cases. The undescended testicle and surrounding adhesions probably retain the viscera and prevent the enlargement of the internal ring and sac.

Umbilical hernias in infants without complications should usually be deferred until the age of 4 to 6 years, as they rarely will result in incarceration or strangulation and may spontaneously close as the child grows older. The rectus muscles constrict the hernial aperature as the child gains age and may finally close the hernial opening. During the early months of life, conservative therapy, such as abdominal strapping and the use of various types of trusses may occasionally cure umbilical hernias. In the repair of an umbilical hernia the umbilicus is not removed. A semicircular incision is made in the crease inside the umbilicus and the skin overlying the hernia sac is dissected free from the sac as well as the surrounding fascia. This is not difficult if ample retraction and careful dissection is done. The sac is dissected down to its neck and excised. A snug peritoneal closure is made at the base of the sac and the surrounding fascia is imbricated with fine silk or cotton sutures. A 'dimpling suture' is placed between the most concave portion of the under side of the umbilical skin and the fascia and tied. This allows the most dependent portion of the umbilicus to be firmly attached to the fascia, preserving its normal concavity. The skin edges are approximated with interrupted nonabsorbable sutures and a pressure dressing is applied after first placing a moistened cotton ball in the umbilicus to maintain postoperative pressure. This pressure is continued during the first week of healing. The skin surrounding the umbilicus is tougher and heals less quickly than other abdominal skin and sutures in this area should be left in place 8 to 10 days to prevent separation of the suture line.

SUMMARY

Since January, 1946, to the present, we have operated upon 60 infants and children under 12 years of age for indirect inguinal hernias and 9 umbilical hernias, by the technics described. In no instance, thus far, have we noted recurrence or testicular atrophy, and there has been no mortality. There have been a few instances of stitch abscesses which were inconsequential. Most of these patients went home the first or second postoperative day and with the changing of diapers and the application of questionable sterile dressings by the family, some redness and drainage from the sutures have occurred. None, however, has had a serious infection. The youngest child in the hernia series was 1 month and the average age was 5 years. The exact number of pre-hospital incarcerations is not known but the percentage was high under 4 years of age.

REFERENCES

1. Coles, J. S.: Operative cure of inguinal hernia in infancy and childhood, Am. J. Surg. *69:* 366 (Sept.) 1945.
2. Duckett, J. W.: Treatment of congenital inguinal hernia, Ann. Surg. *135* No. 6: 879 (June) 1952.
3. Ferguson, A. H.: Oblique inguinal hernia-typic operation for its radical cure, J. A. M. A. *33:* 6 (July) 1899.
4. Herzfeld, G.: Hernia in infancy, Am. J. Surg. *39:* 422 (Feb.) 1938.
5. Potts, W. J., Riker, W. L., and Lewis, J. E.: Treatment of inguinal hernia in infants and children, Ann. Surg. *132:* 566 (Sept.) 1950.
6. Unpublished data.

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HYPERTROPHIC SCARS AND KELOIDS

Excessive scarring and keloid formation following surgery or trauma continue to harass the surgeon. In the minds of the patient and his family, too often these by-products of repair nullify the effects of good surgery by causing surface deformities and disfigurements. Although a temporary or permanent predisposition to proliferative fibroplasia may exist, an understanding of the fundamentals of wound repair will lessen the incidence of these complications. What is not known about the fundamental processes that initiate wound repair and, then, *put on the brakes* when healing reaches a certain point is abyssmal, but some of the fundamental facts about rapid and complete wound healing which lessen scarring are well known.

The known facts relating to the locale of the wound should be kept in mind in all wound care and in planning incisions.

1. The more rapidly a wound heals, the more completely it heals without excessive fibrosis or scar hypertrophy. Healing should not be from the inside out nor from the outside in but should be simultaneous in the depths and on the surface. The only reason for packing a wound open is the prevention of sealed-in infection, blood, serum, or foreign body, or the filling-in of dead space.

2. The blood supply of the part is the most important single factor influencing wound healing, yet this factor, *per se*, is not so important to scar formation.

3. Infection delays wound healing and promotes fibroblastic overgrowth.

4. Foreign bodies in the wound act as a nidus of infection and, by their own presence as well as the infection they incite, produce scar.

5. The factor so important but too often neglected is immobilization, putting the wounded part at rest by splinting, pressure dressing, or other means until adequate healing has occurred. Judgment in the use of such immobilization is required as the prolonged rest treatment of a mobile structure such as a hand may produce a useless appendage.

6. A principle not followed often enough: incisions should be made and/or closed in line with the normal lines of tension (in general, the crease lines) to prevent excessive scar formation and perhaps severe contractures over joints. In certain areas, such as over the deltoid, back of the ear, or over the anterior chest wall, where the skin is under constant tension or motion, enlarged scars occur commonly.

General factors influencing excessive scar formation are far less well known. A racial tendency to keloid formation is most pronounced in the Negro, but it is a well established observation that hypertrophy of scar is seen more often in the young and in people at times of endocrine stress; *e.g.*, both sexes at puberty, females during pregnancy, and patients without thyroid or adrenal dyscrasia. Considerably greater quantities of estrogenic substances than in most other tissues have been assayed in keloids and the infection of estrogens has produced keloids in monkeys. Thin scars of years' durations have been reported to have formed keloids during pregnancy. Young girls, following severe burns, often develop hirsuitism with which may occur changes in the 17-ketosteroid urinary output. Following a severe burn or other stress phenomenon, a patient previously not a keloid-former, as shown by thin flat scars from previous surgery or trauma, may undergo a change which produces massive scar hypertrophy in healed burns and in subsequent surgical scars. There is a variable time factor involved in this *keloiding* tendency. Following healing, the tendency to scar hypertrophy is marked for a few months, after which partial resolution occurs as the healing granulation tissue contracts. The time of this fibroblastic maturation may be much later and, in the severest of *keloid-formers*, may be scarcely evident. Surface wounds, especially from mixed second and third degree burns, allowed to heal slowly without skin grafting, produce the worst hypertrophy of scar.

Experimental work, laboratory and clinical, in regard to the fundamental causative mechanism of keloid formation, has been inconclusive but stimulating. *Keloid-formers* may contain a substance in their serum that promotes *fibroplasia*, especially of the patients' own fibroblasts in tissue culture. The quantitative presence of leukocytes in open wounds may promote fibroblastic proliferation. Some of the endocrine-stimulating or blocking drugs may affect late fibroplasia, but clinical and laboratory trial of ACTH and Cortisone have been disappointing. The enzymatic attack on nucleoprotein of fibroblastic nuclei gives some promise. So-called selective vasoconstrictors, such as kutapressin which has been used to diminish the blood supply of the area undergoing fibroplasia, and hyaluronidase,

the inhibitor of the *glue* of the ground substance, injected locally, have proved nothing of value. The challenge of a very fundamental problem still exists. It has been stated that the fundamental processes initiating wound repair and those inhibiting it are the same for which the answer is sought in the cause of neoplasia. A keloid has been described as a transition stage between neoplastic disease and the inflammatory state, or its descendant.

The treatment or control of hypertrophic scars or keloids is preventative first, and second, reconstructive. Wounds should be healed promptly. The following technical considerations are important for prevention:

1. Remove all dead tissue, secure hemostasis, and use the finest of suture and ligature material to minimize foreign body reaction.

2. Prevent or control infection by surgical technic, the use of antibiotics, and insuring good blood supply.

3. Make incisions and close wounds in the lines of skin tension.

4. Put the part at rest at least during the early healing period. When excessive scarring is evident, the normal sequence of events must be remembered. Early in the hypertrophic stage, rest and relief of tissue tension is helpful, but surgery should not be undertaken until maturation of the scar has started so as not to add more scar on scar. Six months after a wound has healed is the arbitrary time usually set for revision of scars or excision and resurfacing by grafts. Somewhat empirically, roentgen ray or radium therapy has been given, the earlier the better (usually four to seven days), with helpful results in the prevention of further scar hypertrophy. About one-half the cases treated are definitely helped but the dosage must be small and not repeated often for fear of late radiation damage. Massaging or manipulating does not help much; in fact, by preventing rest, they may make things worse.

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The Southeastern Surgical Congress will hold its next meeting in Louisville, Kentucky, March 9, 10, 11, 12, 1953, Brown Hotel.

The following speakers are scheduled to appear on the program:

Dr. Paul P. Salter, Jr., Birmingham, Ala., "Medical Shock".
Dr. John T. Stage, Jacksonville, Fla., "Postoperative Shock".
Dr. Richard S. Wilson, Spartanburg, S. C., "Fluid and Electrolyte Balance in the Surgical Patient".
Dr. Sterling Edwards, Birmingham, Ala., "The Management of Postoperative Pulmonary Bronchial and Vascular Congestion".
Dr. Frank T. Kurzweg, New Orleans, La., "Pulmonary Complications Following Abdominal Surgery".
Dr. Donald Proctor, Baltimore, Md., "Respiration and Anesthesia".
Dr. George H. Yeager, Baltimore, Md., "Postoperative Vascular Complications".
Drs. William H. Prioleau and J. Manly Stallworth, Charleston, S. C., "Gaseous Distention of the Abdomen: Its Significance, Prevention and Treatment".
Dr. Hugh A. Gamble, Greenville, Miss., "Obstructive Enterostomy and the Treatment of Obstruction of the Small Bowel".
Dr. John W. Devine, Jr., Lynchburg, Va., "Duodenal Intubation".
Dr. H. Thurston Whitaker, Vicksburg, Miss., "The Complications of a Wound Following Surgery".
Drs. J. D. Martin, Jr., Emory University, Ga., E. D. Grady and William C. McGarity, Atlanta, Ga., "The Complications of Gastrectomy for Duodenal Ulcer".

SYMPOSIUM—*Postoperative Complications*

Dr. Nathan A. Womack, Chapel Hill, N. C., "The Significance of Benign Lesions of the Breast".
Dr. Guy W. Horsley, Richmond, Va., "The Management of Carcinoma of the Breast".

SYMPOSIUM—*Appendicitis*

Dr. John Mauldin, Atlanta, Ga., "Appendicitis I".
Dr. Richard Smoot, Decatur, Ga., "Appendicitis II".
Dr. Jack Mickley, Hollywood, Fla., "Appendicitis III".
Dr. Brown M. Dobyns, Cleveland, Ohio, "The Management of Carcinoma of the Thyroid".
Dr. Angel Reaud, Havana, Cuba, "Toward Stressing the Friendly Relations Between the Physicians of the United States and Cuba to Our Mutual Advantage".
Dr. Harry Lee Claud, Washington, D. C., Presidential Address
Dr. Arnold S. Jackson, Madison, Wis., "Surgical Lesions of the Neck".
Dr. Richard M. Garrett, Baltimore, Md., "Cholecysto-Cardiac Disease".
Dr. Hu C. Myers, Philippi, W. Va., "Persistence of Pain Following Cholecystectomy".
Dr. Donald S. Daniel, Richmond, Va., "The Question of Drainage Following Cholecystectomy".
Dr. Richard H. Overholt, Brookline, Mass., "The Fourth Dimension in Pulmonary Surgery".
Dr. Frederick F. Boyce, New Orleans, La., "Certain Practical Considerations in the Management of Carcinoma of the Lung".
Dr. Walter J. Burdette, New Orleans, La., "Cardiac Surgery".
Dr. William K. Swann, Knoxville, Tenn., "Surgery of the Patent Ductus".
Dr. G. B. Hodge, Spartanburg, S. C., "Congenital Esophageal Atresia with Tracheoesophageal Fistula".
Dr. Hawley Seiler, Tampa, Fla., "Thoracic Problems".
Dr. Garnet W. Ault, Washington, D. C., "Carcinoma of the Rectum—Factors Responsible for Recurrent or Residual Disease".
Drs. William Clarke Quinn and Alton Ochsner, New Orleans, La., "Bleeding as a Complication of Diverticulosis or Diverticulitis of the Colon".

Dr. Henry W. Mayo, Jr., Charleston, S. C., "Carcinoma of the Stomach—A Challenge to the Physician".
Dr. Max P. Rogers, High Point, N. C., "Para-sympatholytic Blocking Agent".
Dr. Neal Owens, New Orleans, La., "Treatment of Cancer of the Face and Oral Cavity".
Dr. McCarthy DeMere, Memphis, Tenn., "The Role of the General Surgeon in Modern Day Plastic Surgery".
Dr. James A. Harrill, Winston-Salem, N. C., "Indications and Contraindications of Tonsillectomy".
Dr. Branham B. Baughman, Frankfort, Ky., "Hip Fractures in General Surgery".
Dr. Joseph H. Boland, Atlanta, Ga., "Subdeltoid Bursitis".
Dr. Raymond M. Curtis, Baltimore, Md., "The Treatment of Acute Hand Injuries".
Dr. E. L. Gage, Bluefield, W. Va., "Intervertebral Disc Ruptures in Miners".
Dr. William J. Tobin, Washington, D. C., "Repair of the Neglected Ruptured and Severed Tendon Achilles".
Dr. Thomas G. Orr, Jr., Kansas City, Kans., "Urban-Maes Technique of Leg Amputation".
Dr. John C. Burch, Nashville, Tenn., "Hysterectomy—Complications".
Drs. R. B. Chrisman, Jr., and Edward F. Fox, Miami, Fla., "Management of Ectopic Pregnancy".
Drs. Lanier Lukins and J. B. Lukins, Louisville, Ky., "Some Aspects of Endometriosis".
Dr. Amos R. Koontz, Baltimore, Md., "Lantalum Gauze in the Presence of Infection."
Dr. Fred C. Reynolds, St. Louis, Mo., "Treatment of Degenerative Arthritis of the Hip."

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